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HUMAN RESPONSE TO SUSTAINED ACCELERATION

Fraser

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A literature review by
T. M. Fraser, M.Sc., M.D.

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Foreword

This report, prepared under Contract NASr-115, is the first in a series of studies concerned with human response to environmental stress. Its purpose is to provide a critical review of the open literature in the field, and is intended primarily for biomedical scientists and design engineers.

The manuscript was reviewed and evaluated by leaders in the scientific community as well as by the NASA staff. Although there was varied opinion about the author's interpretation of the data compiled, there was nonetheless complete satisfaction with the level and scope of the study. It is anticipated that this study will become a basic building block upon which research and development within the space community may proceed.

JACK BOLLERUD, *Col., USAF, MC*
Deputy Director, Space Medicine
Office of Manned Space Flight

Preface

With a subject such as sustained acceleration, it is manifestly impossible to examine, or in fact even to read, all the papers that have been written, and inevitably some papers of value will have been missed. Apologies are presented to the authors of these. An attempt has been made, however, by reference to definitive work, to present a coherent pattern of response. This study was completed early in 1964, and much work in the field of sustained acceleration has been undertaken since then. Consequently, there are some advances in knowledge not included.

Since some criticism is included in the text, this must be considered in part as a critical review. It is also, however, an appreciation of the current state of our knowledge, as far as it can be ascertained. Any criticism is made humbly, and, it is hoped, objectively. Wherever possible, recommendations are also made on future lines of research.

The format of the paper comprises textual material, figures, and tables. Appropriate recognition, by reference, is given to the origin of the textual material. Figures and tables, with the exception of those obtained from U. S. Government documents, are reprinted by permission of the author and/or publisher.

In a departure from the usual format, an annotated bibliography has been included instead of a list of references.

T. M. F.

Contents

CHAPTER 1

Introduction

> It is a thing of no great difficulty to raise objections against another man's oration,—nay, it is a very easy matter; but to produce a better in its place is a work extremely troublesome.
>
> —*Plutarch:* "Of Hearing. 6"

Outside of the problems associated with reduced barometric pressure, there is no field in the whole of aerospace bioscience that has received so much attention as that of acceleration. In consequence, a plethora of literature has arisen, good, bad, and indifferent, which by sheer volume almost defies analysis on the part of the most persistent reviewer. However, since much of the literature is repetitious or concerned with elucidation of the obvious, it is possible without too much sacrifice to obtain a tolerably clear picture of where we stand today in this field.

For clarity of communication certain explanations and definitions are necessary.

NOMENCLATURE

The descriptive nomenclature used in this paper is the triaxial physiological system proposed by Clark et al.[46] (1961) and illustrated in table 1, which was prepared by Gell[82] (1961). It is based on the inertial displacement of body fluids and viscera with respect to the skeleton when the body, while exposed to acceleration, is positioned with respect to X-, Y-, and Z-axes as shown. Thus, the X-axis is perpendicular to the vertebral column in the anteroposterior direction, the Y-axis is perpendicular in the lateral direction, and the Z-axis is in line with the vertebral column.

The term "G" is used to indicate the inertial resultant of body acceleration, not the acceleration itself. It thus represents the resultant of vehicular displacement acceleration and gravitational acceleration, and is measured in gravitational units, which will be discussed.

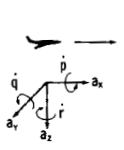
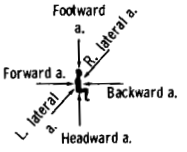
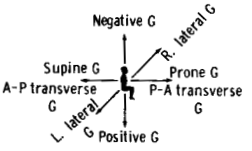
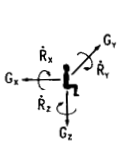
Positive and negative signs are used to delineate the vector. By convention $+G_z$ (previously termed positive G) indicates that the heart is displaced caudally; $-G_z$ (negative G) indicates that the heart is displaced towards the head. Similarly, $+G_x$ (forward acceleration) indicates displacement of the heart toward the back, and $-G_x$ (backward acceleration) indicates displacement of the heart toward the sternum. Again by convention, $+G_y$ (lateral acceleration) indicates displacement of the heart to the left, and $-G_y$ (lateral acceleration) indicates displacement to the right.

A vernacular nomenclature has also arisen, attributed to the astronauts, whereby the inertial resultant is appreciated in terms of the eyeball. Thus, $+G_x$ is eyeballs in (EBI), $-G_x$ is eyeballs out (EBO), $+G_z$ is eyeballs down (EBD), and so on.

The nomenclature for angular acceleration, which will be defined later, is also illustrated in table 1. Thus $+\dot{R}_x$ represents forces about the roll axis that cause the heart to roll to the left, and $-\dot{R}_x$ indicates the opposite direction; $+\dot{R}_y$ refers to forces that produce a downward pitch of the heart, and $-\dot{R}_y$ is applied to the upward direction; $+\dot{R}_z$ indicates a yaw of the heart to the left, and $-\dot{R}_z$ indicates a yaw to the right. Nomenclatures are also discussed by Gell[82] (1961), Dixon and Patterson[57] (1961), Clark et al.[46] (1961), and Gauer and Zuidema[79] (1961).

The terms employed in describing duration

TABLE 1.—*Nomenclature for body acceleration.* [Gell,[82] 1961.]

Direction of motion	Table A Direction of acceleration		Table B Inertial resultant of body acceleration		
	Aircraft computer standard (System 1)	Acceleration descriptive (System 2)	Physiological descriptive [a] (System 3)	Physio'ogical computer standard (System 4)	Vernacular descriptive
	 System 1	 System 2	 System 3	 System 4	
Linear					
Forward	$+a_x$	Forward accel.	[b] Transverse A–P G Supine G Chest-to-back G	$+G_x$	Eyeballs in
Backward	$-a_x$	Backward accel.	[c] Transverse P–A G Prone G Back-to-chest G	$-G_x$	Eyeballs out
Upward	$-a_z$	Headward accel.	Positive G	$+G_z$	Eyeballs down
Downward	$+a_z$	Footward accel.	Negative G	$-G_z$	Eyeballs up
To right	$+a_y$	R. lateral accel.	Left lateral G	$+G_y$	Eyeballs left
To left	$-a_y$	L. lateral accel.	Right lateral G	$-G_y$	Eyeballs right
Angular					
Roll right	$+\dot{p}$		Roll	$-\dot{R}_x$	
Roll left	$-\dot{p}$			$+\dot{R}_x$	
Pitch up	$+\dot{q}$		Pitch	$-\dot{R}_y$	
Pitch down	$-\dot{q}$			$+\dot{R}_y$	
Yaw right	$+\dot{r}$		Yaw	$+\dot{R}_z$	
Yaw left	$-\dot{r}$			$-\dot{R}_z$	

[a] The capital letter G is used as a unit to express inertial resultant to whole-body acceleration in multiples of the magnitude of the acceleration due to gravity. Acceleration due to gravity g_o is 980.665 cm/sec^2 or 32.1739 ft/sec^2.

[b] A–P refers to anterior-posterior.

[c] P–A refers to posterior-anterior.

of acceleration also require examination. Gauer [79] (1961) has defined abrupt acceleration as ranging from 0 to 2 seconds, brief acceleration as ranging from 2.1 to 10 seconds, long-term acceleration from 10.1 to 60 seconds, and prolonged acceleration as anything over 60 seconds.

As will be noted in the future discussion there is a difference in body response to accelerations of duration below and above approximately 0.2 second, related to the latent period for the development of hydrostatic effects. This, then, would appear to be a useful time division and will be considered the limit of abrupt or impact acceleration, rather than the 2 seconds proposed by Gauer. While of necessity all time divisions are arbitrary, it seems unnecessary to subdivide the field as much as Gauer suggests.

Thus, *abrupt acceleration* in this paper will be considered as ranging to 0.2 second duration, *brief acceleration* as ranging to 10 seconds, and *prolonged* as greater than 10 seconds. *Sustained acceleration* will be used to

describe either brief or prolonged acceleration. There is some evidence (Snyder,[157] 1963) that a duration threshold exists still shorter than 0.2 second, where there is insufficient time available for any body reaction to acceleration.

Although sporadic investigation into the effects of acceleration on the body probably began with Erasmus Darwin in 1794, and although various proponents of drastic naturopathy had advocated, and in some cases used, varieties of angular and radial acceleration as therapy for sundry disorders, intensive scientific investigation did not begin until the 1930's. Investigation in the 1930's was chiefly concerned with the examination of certain untoward symptoms of "blackout" and confusion that had been observed among aviators during tight turns. With the advent of World War II and the increase in speed and maneuverability of aircraft, investigation became still more intensive and a large body of knowledge accrued, pertaining particularly to the effect of brief accelerations of the order $\pm 5G_x$.

The demands of space flight, however, have pushed the requirements far beyond that level. To achieve orbital velocity of 18,000 mph requires a total impulse of about 820 G-seconds, while escape velocity of 25,000 mph requires a total impulse of about 1,140 G-seconds. Theoretically the development of the required G-load could be distributed evenly throughout the available time, with a slow onset to less than 2G followed by a corresponding offset. This method would entail only minor physiological problems. Equally, in contemplating travel to distant planets and beyond, the ideal would be to maintain a slow acceleration for half the distance to a readily tolerable maximum, followed by a slow deceleration. In this regard, it is interesting to note that an acceleration of 3G for 1 hour, which has been tolerated (Miller et al.,[128] 1959; Bondurant et al.,[19] 1958), would result in a final velocity of 235,000 mph, nearly 10 times escape velocity. Unfortunately, controlled acceleration of this order is still beyond the capacity of available boosters and is likely to be for the foreseeable future, although development of nuclear and other exotic powerplants will bring this ideal closer. Because of the limitations of current rocket engineering, a compromise is made whereby the necessary total impulse is achieved in stepwise fashion.

Thus, at this juncture, one of our major concerns in the field of acceleration is determining the response and tolerance of man to the forces encountered in present and future space flight. This, however, should not be allowed to blind us to the necessity for a critical reexamination of the problems associated with acceleration in general.

THE NATURE OF ACCELERATION

It is not inappropriate at this time to review some of the fundamental physics of acceleration.

By definition, acceleration is a rate of change, and it can occur in any or all of three related, but differing, maneuvers. Linear acceleration is the rate of change of velocity of mass, the direction of movement of which is kept constant. Thus, as Newton pointed out 300 years ago, acceleration acting on a mass will produce a force, and that force will exert a pressure on the mass, causing it to move if it is movable or to deform if it is not.

Angular acceleration is the rate of change of direction of a mass, the velocity of which is kept constant. In this regard, the acceleration is directly proportional to the square of the velocity and inversely proportional to the radius of the turn. By common usage, where the axis of rotation is external to the body, as in an aircraft turn or a centrifuge, the acceleration is frequently termed "radial" acceleration, while the term "angular" is better retained for situations where the axis of rotation passes through the body.

In its third form, acceleration occurs as a component of the attraction between masses. Newton showed, although he did not finally formulate, that the force of attraction between masses is directly proportional to the product of the masses and indirectly proportional to the square of the distance between them, and that the proportionality constant is the gravitational constant g, which repre-

sents an acceleration of 32.24 feet per second per second when earth's gravitation is a component of the system. This is the accepted unit of measurement of acceleration.

When a mass is acted upon by the acceleration due to gravity, the resulting force, acting vectorially, represents its weight. When the acceleration is other than gravitational, it interacts with gravitational acceleration, if present, to produce a resultant which effectively increases the weight of the mass in the direction of the resultant. It is this effective increase in weight which, in one form or another, is largely responsible for the physiological and other changes found in the body exposed to sustained acceleration.

In this regard, it must be remembered that the body is essentially a fluid system and reacts accordingly. The words of Stapp[159] (1948) are pertinent here:

> To the human engineer, man is a thin flexible sack filled with thirteen gallons of fibrous and gelatinous material, inadequately supported by an articulated bony framework. Surmounting this sack is a bone box filled with gelatinous matter attached to the sack by means of a flexible coupling of bony and fibrous composition. Fuel and lubricants are conveyed to all parts of this machine by flexible hydraulic systems with low pressure tolerances activated by a central pump.

This leads to a brief consideration of hydrostatics. Pascal, in the 17th century, showed that in ideal fluids at rest, (a) fluid pressure is equal in all directions, (b) pressures at points lying in the same horizontal plane are equal, and (c) pressure increases with depth under the free surface. This increase is equal to ρ gh dynes/cm^2, where ρ is the density of the fluid, g the gravitational constant, and h the depth.

These laws apply to the vascular system and, after a fashion, to the body as a whole. Thus, a mean arterial pressure at heart level of 100 mm Hg will support a column of blood 130 cm high (Burton,[30] 1960), and the mean arterial pressures at feet and brain are approximately the same when the body is horizontal. When the body is erect, the effect of change in weight is such as to decrease mean arterial pressure in the brain and increase it in the feet. Thus, if in the erect position the brain is 25 cm above the heart, the mean arterial pressure becomes $100 - (250/13)$, or 81 mm Hg. Similarly, arterial pressure in the feet will become approximately 175 mm Hg.

Under increased acceleration, the resultant between the applied and the gravitational acceleration acts to increase the effective density of the blood or, in other words, increases the g component in the ρ gh equation according to the magnitude of g units of the applied acceleration. This is illustrated in figure 1 (Wood et al.,[193] 1963), which is a diagrammatic representation of hydrostatic pressures in the vascular system of a man sitting in the upright position at 1G and under exposure to $+5G_z$. The center sketch, illustrating the vascular effects at 1G, indicates that with a mean arterial pressure of 120 mm Hg the mean pressures at head and foot levels are calculated to be 96 and 170 mm Hg, respectively. At $+5G_z$, while maintaining mean arterial pressure at heart level of

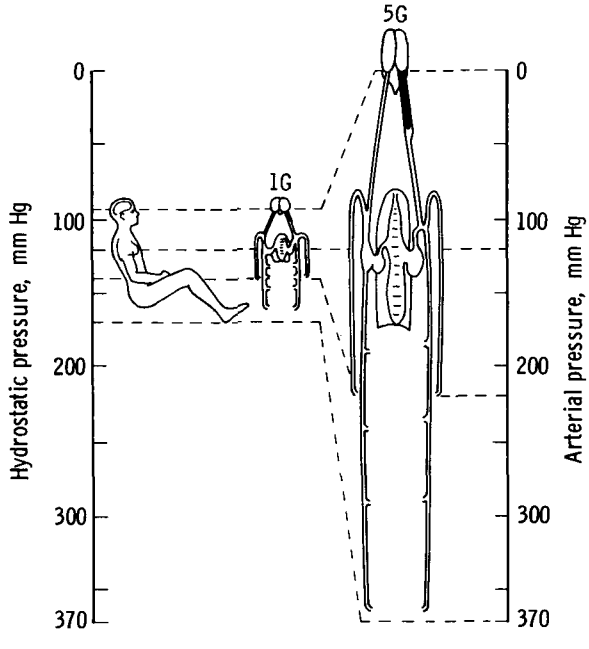


FIGURE 1.—*Diagrammatic representation of hydrostatic pressures in vascular system of a man in upright sitting position at 1G and during headward acceleration at 5G.* (Wood et al.,[193] 1963.)

120 mm Hg, the calculated pressure at the base of the brain will be zero, while at the feet it will be 370 mm Hg. Under these circumstances the subject would be unconscious, and an additional venous pressure of 250 mm Hg would be required to return blood from feet to heart. It will be shown later that unconsciousness does not necessarily occur at $+5G_z$, since compensatory adjustments occur.

The same hydrostatic principles apply in the venous system, although the effect is modified by the action of valves.

FACTORS IN RESPONSE TO SUSTAINED ACCELERATION

In assessing the acceleration stress involved, several factors must be specified for full delineation. Of these, the five major variables are:

(a) Magnitude of the peak or peaks of acceleration

(b) Duration of the peak or peaks of acceleration

(c) Total duration of the acceleration from time of onset to completion of offset

(d) Direction of the primary or resultant acceleration with respect to the body axes

(e) Rate of onset and offset

It is difficult, if not impossible, to provide an expression or multidimensional graph covering all these variables. Consequently, any definition of an acceleration stress tends to be limited in its application or highly complex in its delineation.

Still other variables complicate the picture. Chambers [35] (1963) has listed 10 of these as follows:

(a) Types of end points used in determining tolerance

(b) Types of G-protection devices and body restraints used

(c) Body position, including specific back, head, and leg angles

(d) Environmental conditions such as temperature, ambient pressure, and lighting

(e) Age of subject

(f) Emotional factors such as fear and anxiety, confidence in self and apparatus, and willingness to tolerate discomfort and pain

(g) Motivational factors such as competitive attitude, desire to be selected for a particular space project, or specific pay, recognition, or awards

(h) Previous acceleration training and accumulative effects

(i) Techniques of breathing, straining, and muscular control

(j) Presence or absence of performance tasks which must be done during peak G stress

In addition, the effects of acceleration stress are modified by:

(a) Coupling between the individual and the vehicle of application (seat, couch, etc.)

(b) Anthropomorphic form of the individual

(c) Physical characteristics (*e.g.*, impedance) of the body and its components which modify the transmission of force

Further, it must be remembered that most experimental work in acceleration stress, with some notable exceptions, has been carried out with very few subjects, who may or may not represent an evenly distributed selection from the curve of normal variation.

CHAPTER 2

The Natural History of Sustained Acceleration Stress

In the operational situation it is unusual, if not impossible, for acceleration stress to occur in a simple form. One is rarely if ever exposed, for example, to a simple unvarying $+G_z$ stress. Instead, acceleration stress may vary in its resultant vector, magnitude, and type, and may be accompanied by complex oscillations and vibrations. For purposes of analysis at this time, however, it is simpler to consider the response to a continuous resultant vector without additional complexities. This is largely achieved by use of a centrifuge. It will be noted, however, that because of the physical motion applied by a centrifuge, initial tangential acceleration will be imparted at onset of the acceleration, so that during the buildup of angular velocity the subject is exposed to two imposed acceleration fields plus gravitational acceleration.

Although it has been realized since the beginning of centrifuge experimentation that the occurrence of concomitant tangential acceleration might modify the human response to the applied radial acceleration, the resulting problems have generally been considered negligible and have never been fully investigated. In addition, at least one human centrifuge imparts an added angular acceleration to applied radial acceleration as the gondola rotates into its optimum position.

Before considering the natural history of acceleration exposure on a centrifuge it is wise to examine the effectiveness of a centrifuge in simulating the expected stress. To determine this, Lambert[108] (1946), using a specially instrumented aircraft, investigated the responses of 42 men exposed to positive accelerations of 4 to $5G_z$ for 10 to 15 seconds and compared their tolerances and certain physiological changes with those found under similar circumstances on the Mayo centrifuge. He found that the tolerance of subjects as pilots was 0.7G higher than when they were exposed as passengers, and 1.4G higher than as subjects in the centrifuge. Compensatory changes were the same but tended to occur a few seconds sooner in the air than in the centrifuge.

Lambert suggested that the factors modifying the results included the emotional tension of flying, a colder environment, and acquired protective maneuvers on the part of the pilots in flight, but concluded, with justification, that the human centrifuge is a valid means for studying the physiological effects of acceleration. No other comparative study has been carried out, but it appears that the centrifuge provides a reasonable verisimilitude, although it is probable that the threshold figures obtained by centrifuge experimentation represent lower levels of tolerance and performance than occur operationally.

With this in mind, the natural history of exposure to sustained acceleration will be described at this time without delineating or interpreting the physiological effects, tolerance thresholds, and performance capacities. These are discussed fully in subsequent sections.

POSITIVE ACCELERATION ($+G_z$)

It is frequently overlooked that man in a terrestrial environment is continuously exposed to $1G_z$, which fortunately he normally withstands without incident. Occasionally, however, as in prolonged standing erect, hydrostatic effects manifest themselves in syncope. This sometimes has aeromedical sig-

nificance when it occurs in an individual rigidly strapped in a seat in such a manner that he cannot fall to the horizontal and consequently reduce the heart-brain distance.

The sensations and symptoms that occur as a result of positive and negative acceleration have been described by several authors: Armstrong and Heim,[6] 1938; Armstrong,[5] 1939; Ruff and Strughold,[142] 1939; Ham,[90] 1943; Franks et al.,[71, 72] 1945; Lambert and Wood,[109] 1946; Gauer,[78] 1950; Ryan et al.,[144] 1950.

With slow increase in magnitude toward 2G, an increase in weight is observed, with increased pressure on the buttocks in the seated position and drooping of the soft tissues of face and body. By 2½G it is nearly impossible to raise oneself, and by 4G the arms and legs can hardly be lifted.

Hydrostatic effects manifest themselves in the relaxed unprotected subject in the seated position after about 3 seconds' exposure to 3 or 4G, with progressive dimming (grayout) of peripheral vision. Tunneling of vision occurs at 3½ to 4G and complete loss of vision (blackout) at 4½ to 5G after a total plateau exposure of about 5 seconds. Hearing and consciousness are retained for a few seconds longer but are finally lost. In 50% of subjects, mild to severe convulsions occur during the unconscious period, and recovery (assuming the stress is immediately reduced) is frequently accompanied by bizarre dreams. Blackout and unconsciousness are sometimes associated with paresthesias, confused states, and, more rarely, gustatory sensations. No incontinence has been observed. During the onset, passive and compensatory physiological changes take place which will be discussed. Pain is not normally a feature, but the lower portions of the legs feel congested and tense; there may be muscular cramps and tingling. Inspiration becomes difficult, and eventually the subject exhibits a tendency to hold his breath in the mid-inspiratory position. Reaction times are prolonged and task performance is reduced even before the level of unconsciousness. If unconsciousness occurs, a loss of orientation for time and space persists for about 15 seconds after cessation of acceleration.

NEGATIVE ACCELERATION ($-G_z$)

With application of negative acceleration ($-G_z$) in the unprotected subject there is a feeling of facial suffusion and cranial fullness which is tolerable but unpleasant. This is accompanied by reflex cardiovascular changes which will be discussed. Increasing the magnitude to between $-2G_z$ and $-3G_z$ produces considerable facial congestion and throbbing headache. At about $-3G_z$ for 5 seconds, blurring and graying of the vision occurs and in some subjects there is a reddening of the visual field of debatable origin. A few individuals, with practice, may tolerate up to $-5G_z$ for 5 seconds in the unprotected state. On cessation of acceleration, the congestion disappears slowly and may leave petechial hemorrhages, congested and hemorrhagic conjunctivae, and edematous eyelids.

FORWARD ACCELERATION ($+G_x$)

While most of the early work was concerned with the effects of positive and negative acceleration, some of the earlier investigators, such as Bührlen[28] (1937), Gauer[77] (1938), Gauer and Ruff[81] (1939), and Armstrong and Heim[6] (1938), provided descriptions of the response to transverse acceleration. These were supplemented by a multitude of published reports during and since World War II, when it was appreciated that tolerance in this vector was such as to permit the accelerations of rocket launch. Of these, noteworthy reports were prepared by Chambers[34] (1961), Chambers and Hitchcock[36] (1963), Smedal et al.[154] (1960), Bondurant et al.[19] (1958), Clark et al.[44] (1959), and Clark[42] (1961).

Application of up to $+3G_x$ for about 2 minutes to a subject restrained in a contour couch will produce little effect other than a feeling of increased weight and pressure on chest and abdomen with a developing fatigue. At about 3G a slight difficulty in focusing may be observed along with slight spatial disorientation, each of which subsides with experience. In performance tasks initially

there may even be some improvement. However, approaching $+6G_x$ there is a development of tightness in the chest, mild chest pain, some loss of peripheral vision, difficulty in breathing and speaking, decrease in depth of visual field, blurring of vision, and additional effort required in maintaining focus. In control performance tasks there is a tendency to overcontrol.

Toward $+9G_x$, chest pains and pressure become more severe. Breathing is difficult, requiring tensing of chest and stomach, and shallow respiration from a position of nearly full inspiration. Peripheral vision is further reduced, with increased blurring, occasional tunneling, and greater concentration required to maintain focus. Occasional tears are observed. In control performance tasks there is a loss of feel, a tendency to make inadvertent control inputs, and hesitation in making control inputs because of the possibility of inadvertent action.

By $+12G_x$ breathing difficulty is severe, with chest pain and marked fatigue. Peripheral vision is lost and central acuity diminished, with lacrimation. Control is very difficult and requires great concentration.

At $+15G_x$, some subjects report a recurrent complete loss of vision with extreme difficulty in breathing and speaking, loss of sense and feel, and extreme difficulty in control tasks. The pain experienced, when severe, is a gripping viselike sensation around the chest, and is also encountered in severe vertical sinusoidal vibration. Its origin is debatable but it is generally considered to arise from tissue stretching, or perhaps intercostal muscular spasm.

Petechiae of the back and antecubital fossae occur regularly above $+6G_x$, and reflex cardiovascular changes and inertial pulmonary changes are observed which will be discussed later.

On cessation of acceleration the ensuing disability is variable and includes an unsteady gait, dizziness, and occasional nausea, which may persist from 1 to 5 minutes.

BACKWARD ACCELERATION ($-G_x$)

Less work has been done on the effects of backward acceleration. Papers by Clarke et al.[47, 48] (1958, 1959), Bondurant et al.[19] (1958), Smedal et al.[154, 155] (1960, 1963), and Creer et al.[54] (1962) provide some of the later descriptions, although the position was investigated by some of the earlier workers (Gauer,[78] 1950).

In principle, the effects are similar to those of forward acceleration ($+G_x$), with modifications produced by the reversed direction of the vector. Thus in $-G_x$ the chest pressure is reversed and consequently respiration is easier than in $+G_x$. However, since pressure is outward toward the restraint harness, pain and discomfort from pressure on the harness become severe at about $-8G_x$ in the optimal position. Should the head be allowed to tilt forward, hydrostatic effects on the cerebral circulation become manifest at even lower intensities. Another major feature of $-G_x$ acceleration is interference with vision. Despite the greater respiratory comfort the $-G_x$ vector is disliked by operators, perhaps because of a feeling of insecurity engendered by inadequacies of restraint systems.

LATERAL ACCELERATION ($\pm G_y$)

Very little work has been done on the effects of lateral accelerations. The only papers of substance appear to be those of Clark[42] (1961) and Headley[91] (1961). Clark showed that at $3G_y$ for 10 seconds lateral loads become uncomfortable, with pressure on the restraint system and a feeling of supporting the entire weight on the clavicle. This is accompanied by a movement of the hips and legs, and a yawing and rotation of the head toward the shoulder. Pressure effects were found to give rise to petechiae and bruising over the affected clavicles and, in the case of one subject at $-5G_y$ for 2 seconds during a total exposure of 14.5 seconds, to external hemorrhage and severe postrun headache. Headley found, in addition, severe vascular engorgement with pain in the dependent forearm and elbow.

CHAPTER 3

Physiological Effects of Sustained Acceleration

As stated earlier, the physiological changes found in acceleration of brief and longer duration are fundamentally related to the increase in weight and hydrostatic pressure that develops along the vector of the acceleration. Thus the effects vary according to the position of the body with respect to the vector. When applied in the G_z vector the limiting effects are primarily cardiovascular; when applied in the G_x vector, however, they are primarily respiratory; insufficient is yet known about the G_y vector to make a generalization. Because of these differences, each vector will be treated separately.

POSITIVE ACCELERATION ($+G_z$)

The early work on the physiological effects of positive acceleration is ably summarized by Ham [90] (1943) in a comprehensive review covering the work, particularly that of the German investigators, prior to World War II. During World War II the Canadian group and the Mayo Foundation group produced more definitive findings. In particular, Franks et al.[72] (1945) and Wood et al.[190] (1946) showed the sequence and cause of events resulting from exposure to $+G_z$ acceleration.

Blood Pressure

These early findings have been amplified further in an excellent paper by Lindberg and Wood [117] (1963), who point out that during acceleration with rates of onset from 1 to 2G per second, which are of magnitude sufficient to produce loss of vision, there is an immediate decrease in blood pressure at head level, an increase in heart rate, a decrease in blood content of the ear, and a decrease in the amplitude of the arterial pulse at the level of the ear. This leads in turn to failure of peripheral vision and eventually of central vision.

These findings are illustrated in figure 2. It will be noted that recovery of blood pressure and ear pulse begins before the acceleration episode is completed. This rise in pressure is considered to be due to pressor reflexes initiated by the fall in arterial pressure in the carotid sinus, and is consistent with the early observation of Code et al.[51] (1945), who pointed out a recognizable division between the period of progressive failure beginning with the onset of acceleration and the period of compensation which becomes effective 6 to 11 seconds after onset of acceleration. Leverett and Clarke [111] (1959) demonstrated an interesting confirmation of this response by implanting a strain gage in the wall of the left ventricle of dogs and exposing them to 3G for 15 seconds at two different rates of onset, 0.75G per second and 1G per 10 seconds. With the onset of acceleration the investigators observed an initial decrease in both arterial pressure and contraction force. Six to 10 seconds later the force increased to 25% above control, probably contributing to an associated rise in arterial pressure. Since this pressor response is blocked by tetraethylammonium chloride, G. E. Brown et al.[23] (1949) consider that it is mediated by the autonomic system. Lambert and Wood [109] (1946) showed by progressive exposure of subjects to increased magnitude of acceleration that the fall in blood pressure at head level is proportional to the magnitude of acceleration. The blood pressure at heart level, however, is maintained near normal, or in fact may actually increase

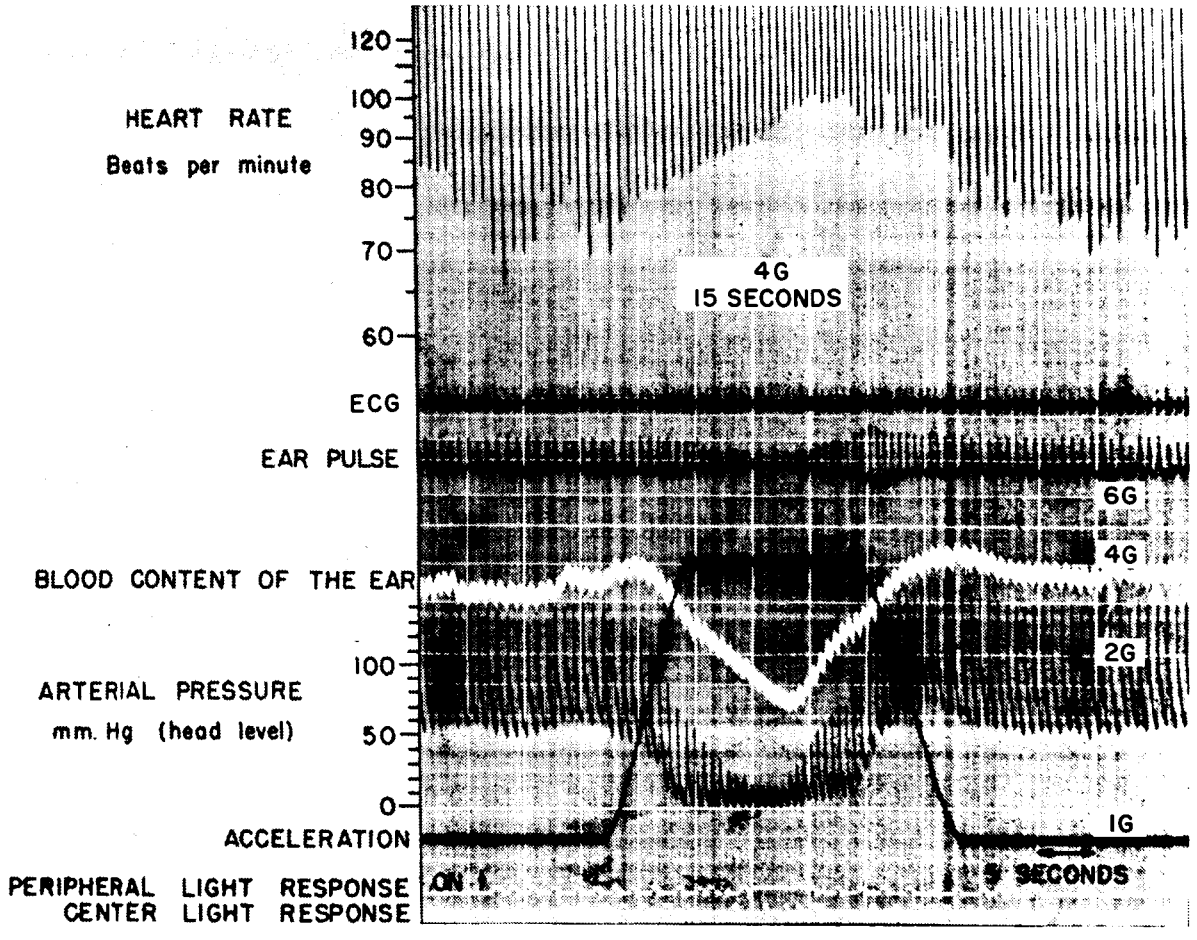


FIGURE 2.—*Sequence of physiological events during exposure of a normal subject to 4G headward acceleration for 15 seconds on a human centrifuge.* (Lindberg and Wood,[117] 1963.)

to hypertensive levels at 5G because of vasoconstrictive compensatory measures.

Venous Pooling

That pooling of venous blood occurs was early observed by Bührlen[28] (1937) who, with a plethysmograph on one leg, found that the volume increased by as much as 350 cc at 10.8G. (In the book *German Aviation Medicine, World War II* that figure is quoted erroneously as being 35 cc.) In addition, pooling of blood takes place in the splanchnic region, although less than might be expected because of the increased hydrostatic pressure in the abdomen and the descent of the diaphragm. Hershgold and Steiner[95] (1960) suggested that this pooling may be aggravated by obstruction to the venous entry to the right atrium because of downward displacement and lengthening of the heart. Because of the anatomical position of the entry into the right atrium, the fixation of the heart, and the fact that the actual descent is less than 1 cm, this seems unlikely.

Cardiac Output

Regardless of the mechanisms, however, there is potential for considerable loss of venous return. This, in turn, gives rise to a decrease in cardiac output, first measured by Howard[98] (1959) in two subjects, using the direct Fick principle. A serious problem in the use of the direct Fick principle is the fact that steady-state conditions are required dur-

ing the experiment for a period long enough to measure a significant oxygen uptake. Steady-state conditions are difficult to achieve under positive acceleration, as indeed are the techniques of measurement.

Consequently, Wood et al.[195] (1961), using highly sophisticated techniques, employed an indicator dilution method whereby sudden single doses of an indocyanine dye were automatically injected via a catheter into the right atrium. The resulting dilution curves were recorded by way of a cuvette oximeter and the cardiac output was calculated. Other determinations included heart rate, ECG, stroke volume, aortic, atrial, and radial artery pressure, systemic vascular resistance, respiratory rate, and intrathoracic (intraesophageal) pressure. To standardize the response of the pressure transducers, an ingenious system was used whereby on completion of recording all were physically referenced to the third intercostal space. A typical photokymographic record is shown in figure 3.

With a rate of onset of about 1G per second, the subjects were exposed in the seated position to levels of 2, 3, and $4G_z$ for plateaus of 60 seconds. A dye dilution curve was recorded 20 to 40 seconds after onset of acceleration since the technique demands a stable blood flow during acquisition of the curves, and most of cardiovascular compensatory reactions occur during the first 15 seconds of exposure to acceleration. The results, which were widely variable, are given in table 2.

The authors themselves anticipated possible criticisms of their work and convincingly justify their approach. They point out the accepted correlation that exists between the dye dilution technique and the Fick method, but note that in an acceleration study stagnant retention of the dye might

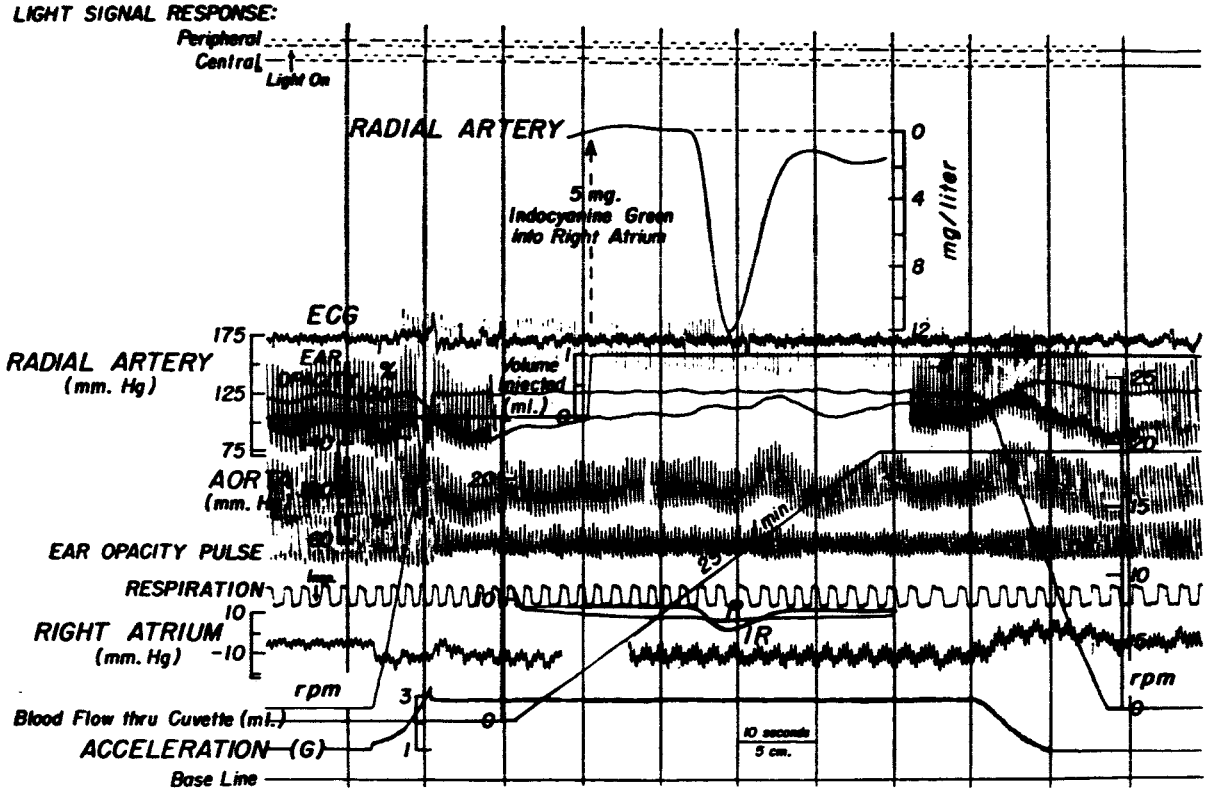


FIGURE 3.—*Typical photokymographic record of indicator-dilution curve and other variables during exposure of a healthy man to headward acceleration of 3G for 70 seconds.* (Lindberg et al.,[116] 1960.)

TABLE 2.—*Changes in cardiovascular quantities during positive acceleration, as compared with control values.* [Wood et al.,[195] 1961.]

Quantity	Percentage increase (+) or decrease (−) at—		
	$+2G_z$	$+3G_z$	$+4G_z$
Cardiac output	−7	−18	−22
Stroke volume	−24	−37	−49
Heart rate	+14	+35	+56
Mean aortic pressure	+9	+21	+27
Systemic vascular resistance	+17	+41	+59

occur. They show, however, that the slopes and appearance of the dilution curves during acceleration were similar to those of the control curves except for changes consistent with decrease in cardiac output, and argue that any retention of dye was therefore insignificant. The argument seems valid although it is not accepted in all quarters.

They examined the question of using the third intercostal space as a pressure reference, bearing in mind that under applied G_z acceleration the heart moves downwards within the chest and no longer bears the same relationship to exterior reference points as it does at $1G_z$. To demonstrate that, in fact, the third intercostal space was a valid reference point under acceleration, they measured the distance between this point and the eye, and calculated that at $4G_z$ a pressure of 112 to 128 mm Hg would be required at the level of this space to prevent the occurrence of blackout; since the pressure actually measured during $4G_z$ was of this order and blackout did not occur, the reference point was considered to be valid.

A similar but less comprehensive study was carried out by Hershgold and Steiner[95] (1960), who exposed dogs in a fully supine position to levels of $+3G_z$ and $+4G_z$ at a rate of onset of 1G per 15 seconds. Determinations of cardiac output were made after 1 minute by means of an indicator dye technique. Results showed decreases in cardiac output and stroke volume of 52% and 65%, respectively, and increases in heart rate and peripheral resistance of 35% and 70%. Although of the same order of change, the greater magnitude in the case of the dogs may be related to the fact that the dogs, although in a G_z vector, were fully supine, while the human subjects exposed to the same vector were seated. Since dogs are normally habituated to a $+G_x$ position their response might be expected to be greater.

Hydrostatic Effects on Vision

Vision is markedly affected by alteration in hydrostatic pressure, and the symptoms manifest themselves at levels below those producing unconsciousness. The explanation was first propounded by Andina[4] (1937), who pointed out that intraocular pressure is some 20 mm Hg higher than intracerebral pressure and consequently blood supply to the retina fails before failure of the cerebral circulation. As confirmation, Lambert[107] (1945) showed that application of 30 to 40 mm Hg negative pressure to the eyeball by means of suction goggles will raise the blackout threshold. Similarly, application of positive pressure will lower the threshold. Livingston[119] (1939) pointed out that the retina has an oxygen requirement four times as great as that of any other body tissue, although he does not specify his standard of comparison. Hence, even a minor reduction of blood supply should cause marked interference with vision.

With remarkable perseverance on the part of the investigator and his assistants, Duane[58] (1954) examined and obtained illustrations of the retina at G_z accelerations to blackout level, and measured arterial pressure in the radial artery with the arm so placed that the tip of the cannula was at eye level. A correlation between visual change and change in the fundus oculi was established as follows:

Stage	*Subjective*	*Objective*
I	Loss of peripheral vision	Arteriolar pulsation—*i.e.*, recurrent exsanguination
II	Blackout	Arteriolar exsanguination and collapse
III	Return of central and peripheral vision	Return of arteriolar pulsation and temporary venous distension

Based on the fact that the inner retinal layers are sensitive to hypoxia it was concluded that retinal arteriolar ischemia produced hypoxia of these layers, and it was later shown (Lewis and Duane,[114] 1956) that the critical site of hypoxia was the junction of the ganglion and bipolar cells in the retina.

In a later paper Duane et al.[60] (1962) showed that where the hydrostatic pressure was such as to cause collapse of the arteriolar vessels during diastole and recovery in systole, a pulsation of the vessels may be observed which is associated with grayout, or reduction of the visual field to approximately 15° in all meridians.

In addition, in those subjects in whom a photic drive of the EEG was observable at rest, loss of photic drive could be demonstrated at grayout level.

Further visual phenomena will be considered later in connection with performance studies.

Unconsciousness

Consciousness is usually lost in a 1G environment when the mean cerebral arterial pressure falls to 25 mm Hg. However, under acceleration sufficient to produce blackout, pressure at eye level is less than 20 mm Hg, yet consciousness may still be maintained. With sufficient magnitude of G-time, however, unconsciousness from cerebral hypoxia will result. The cerebral circulation is discussed by Livingston[120] (1960), who points out the so-called Monroe-Kellie doctrine in which it is stated that because the chamber of the cranial vault and spinal column form a semirigid container with practically incompressible tissue and fluid content, it offers great resistance to change in volume of any single constituent. An equal and opposite effect must occur in one or another of the remaining components.

Because of this and because of the continuity between the venous column and the craniospinal veins, the cerebrospinal fluid helps to preserve an arteriovenous siphon even during acceleration. Thus under $+G_z$ acceleration there is a proportionate fall in arterial and venous blood pressure, but at the same time a decrease in cerebrovascular resistance because of a concomitant decrease in cerebrospinal fluid pressure. While the change in hydrostatic pressure militates against arterial flow, it encourages venous flow and maintains a siphon effect.

Henry et al.[92] (1951) investigated three subjects under accelerations of $+1G_z$ to $+4\frac{1}{2}G_z$ for durations of 1 to 2 minutes and found, among other things, venous pressures in the jugular bulb ranging from 20 to 60 mm Hg below arterial. A siphon effect of this magnitude would of course assist in maintaining circulation. When the magnitude of acceleration is great enough, however—for example, $+6G_z$ for 5 seconds—cerebral circulation becomes inadequate despite maintenance of pressure at heart level, and cerebral hypoxia results. It would appear, however, that cerebral circulation can fail in another manner. In this regard, Leverett and Clarke[111] (1959), with their strain gages implanted in dogs' left ventricles, showed that when 3 to 4G was maintained for 10 minutes, the initial response was one of increased arterial pressure and contraction force, but a subsequent fall occurred between 3 and 7 minutes after reaching peak pressure. Although 3G has been endured for 60 minutes and 4G for 20 minutes (Bondurant,[18] 1958), it is interesting to note that Wood et al.[195] (1961) observed such vasovagal attacks in some subjects who, while exposed to $3\frac{1}{2}G$ for 10 minutes, had had repeated arterial assaults during cardiac output determinations.

It would thus appear that two types of unconsciousness can result, one associated with hypertension at heart level but inadequate tension at eye level, and the other associated with failure of compensation, hypotension at heart level, and syncope. It would seem, however, that the relationship between these two types of unconsciousness under acceleration has never been investigated.

Also deserving of investigation in this regard is the onset of convulsions with unconsciousness. Gauer, writing in *German Aviation Medicine, World War II*, mentions the occurrence of convulsions during uncon-

sciousness. These were also observed and photographed by Franks et al.[72] (1945), who found convulsions and EEG changes in 52% of 230 subjects in 40% of 591 tests producing unconsciousness. These were usually slight clonic seizures involving all or some of the extremities, face, and trunk. Less commonly, severe generalized convulsions were observed, terminating in 2 to 5 seconds. Incontinence was never observed. In later years Sem-Jacobsen[146-149] (1958–1961) found marked EEG changes in pilots undergoing violent flying maneuvers and photographed convulsive episodes in flight. Brent et al.[20] (1960) found that suitable combinations of hyperventilation, hypoglycemia, and $+G_z$ acceleration would produce convulsive episodes in centrifuged subjects. Why some individuals should experience convulsions and others not has never been fully examined. Whether this is due simply to vague "individual variation" or whether there is some other factor related to cardiovascular dynamics is worthy of exploration, as is the relationship between clinical epilepsy and the convulsive episodes associated with environmental stresses such as $+G_z$ acceleration and hypoxia.

Squires et al.[158] (1964) have recently analyzed the EEG changes in human subjects during blackout produced by positive (G_z) acceleration. They exposed 13 different subjects on two separate occasions to $+6G_z$ and to $+7G_z$ at a relatively slow rate of onset. A frequency analysis was made of a one-channel EEG and characteristic changes were noted during grayout and blackout. An increase in beta frequencies (16 to 36 cps) occurred, and the general amplitude pattern was coincident with the acceleration profile. However, the lower end of the beta frequencies (16 to 19 cps) had a tendency to decrease or flatten out during blackout, although at the same time there was an increase in still lower frequencies (5 to 7 cps). An increase in alpha frequencies (8 to 13 cps) often appeared during grayout and blackout, but disappeared when bursts of high-amplitude lower frequency components appeared. The authors considered that the best index of consciousness appeared to lie in this inverse relationship between amplitude of components in the 5-cps range and the depth of blackout.

Arterial Oxygen Saturation

While the major part of the cerebral hypoxia that ensues under positive acceleration is no doubt due to inadequacy of the blood flow, Barr et al.[12] (1959) demonstrated that prolonged positive acceleration could produce marked arterial hypoxemia in anesthetized dogs even when the animals were hyperventilating on 100% oxygen. Lindberg and his colleagues[116] (1960), during their cardiac output studies, measured arterial oxygen saturation during the first 15 seconds of acceleration before injection of the indocyanine dye. After injection, of course, the cuvette was used for readings of dye dilution. They observed a decrease from 97% at 1G to 93% at 4G.

Barr[10] (1962), in a comprehensive set of experiments designed to investigate this situation, found that (a) arterial unsaturation developed during prolonged exposures to positive ($+G_z$) acceleration despite an accompanying considerable increase in respiratory minute volume, (b) exposure to several consecutive episodes increased the rate and degree of unsaturation, and (c) the arterial pH remained relatively unchanged (table 3 and fig. 4).

Since the hypoxemia took place despite hyperventilation with 100% oxygen, it is assumed that an arteriovenous shunt occurred by reason of disturbed ventilation-perfusion relationships. This will be discussed further in connection with the effect of transverse ($\pm G_x$) acceleration. In a later paper Barr[11] (1963) showed that arterial saturation during a 2-minute exposure to $5G_z$ dropped from a mean of 96.2% to 87.4% while the alveolar oxygen tension fell to a mean of 58.0 mm Hg.

Barr comments on the stability of the arterial pH. He points out that the venous admixture would increase arterial P_{CO_2} and tend to veer the pH towards the acidotic; at the same time, increase in acidity secondary to increased CO_2 production and formation of acid metabolites from increased work load under high G would tend to shift the pH still

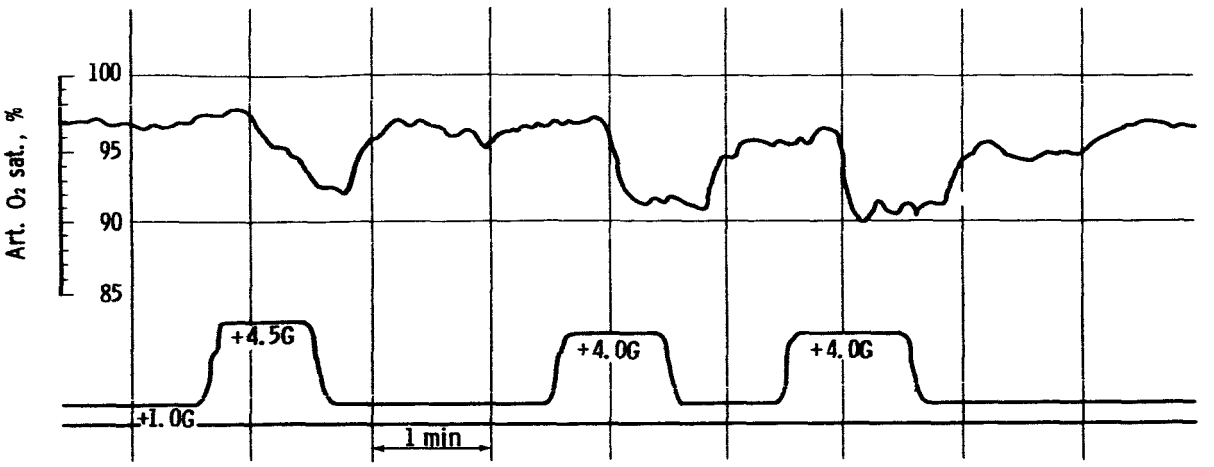


FIGURE 4.—*Time course of changes in arterial oxygen saturation induced by consecutive exposures to positive acceleration (4.5, 4.0, and 4.0G) in subject 20 years old, height 179 cm, weight 68 kg, breathing air and wearing automatically inflated anti-G suit. Note increasing rate and degree of unsaturation in consecutive runs; also impairment of resaturation in the postrun periods.* (Barr,[10] 1962.)

TABLE 3.—*Effect of positive ($+G_z$) acceleration on arterial saturation while wearing anti-G suit and breathing air.* [Barr,[10] 1962.]

Subject			Acceleration, G	Duration of run, sec	Cond. (a)	$\dot{V}_I$, l/min ATPS	Art. O_2 satur., %	Art. pH	Minimum O_2 saturation during run, %
Age, yr	Length, cm	Weight, kg							
17	175	71	+4.5	163	A	11.8	97.3	7.41	89.6
					B	20.4	90.7	7.42	
					B−A	+8.6	−6.6	+0.01	
28	176	72	+4.5	153	A	7.3	97.0	7.44	89.0
					B	16.1	90.0	7.44	
					B−A	+10.2	−7.0	0	
22	166	64	+4.5	122	A	11.9	97.8	7.36	90.5
					B	25.9	94.8	7.37	
					B−A	+14.0	−3.0	+0.01	
24	182	70	+4.5	97	A	11.5	98.6	7.43	94.5
					B	29.8	95.0	7.44	
					B−A	+18.3	−3.6	+0.01	
21	175	72	+4.5	71	A	12.2	96.2	7.43	87.2
					B	19.3	88.0	7.44	
					B−A	+7.1	−8.2	+0.01	
21	175	65	+5.0	74	A	8.4	96.2	7.38	90.4
					B	13.8	91.0	7.39	
					B−A	+5.4	−5.2	+0.01	
24	173	73	+5.0	82	A	11.0	97.0	7.45	79.0
					B	20.0	81.0	7.46	
					B−A	+9.0	−16.0	+0.01	

[a] Condition A: control values during 60 sec immediately prior to run (means for arterial O_2 saturation and pH).

Condition B: values after 60 sec had elapsed from onset of run ($\dot{V}_I$ refers to the first 60 sec after onset of run).

[b] Arterial O_2 saturation and pH values refer to the time when the arterialized blood left the lungs (corrections made for time lags in the readings).

further in the acid direction. Thus, the alkalosis produced by hyperpnea would be balanced and the pH remain unchanged.

Respiration

There has been little investigation of the effect of positive acceleration ($+G_z$) on respiration. The early workers (von Diringshofen,[56] 1934; Gauer,[77] 1938) showed that respiration rate and tidal volume increase between $+1G_z$ and about $+5G_z$. This was again demonstrated by Lombard et al.[121] (1948), who found that positive acceleration up to $+5G_z$ without artificial protection increases the respiratory rate and tidal volume, but decreases the vital capacity. The latter they attributed to a limitation in inhalation imposed by downward pressure on the thorax. Bondurant[18] (1958) investigated the mechanics of breathing under acceleration, measuring the pulmonary compliance in five subjects during accelerations of 3 and $3.5G_z$. He showed a significant decrease in pulmonary compliance and a small increase in functional residual capacity (100 to 600 cc).

Glaister[84] (1961) went a stage further and showed that the diaphragm was pulled down by the weight of the abdominal viscera, potentially increasing the vital capacity by 500 cc, but because of the factors noted above the actual vital capacity diminished by 300 cc. By continuous recording of lung volume and pressure gradient he showed that neither lung stiffness nor air resistance varied, at least to $+3G_z$, but that the total work of breathing increased because of chest wall stiffness and increased intra-abdominal pressure. Thus the overall pulmonary efficiency fell.

Barr[10] (1962) confirmed the lowered pulmonary efficiency. His records, which unfortunately are not suitable for further reproduction, show that with subjects at $+5G_z$ for 1 minute (wearing a G-suit) there is an initial apnea for a few seconds with onset of acceleration, followed by a marked increase in respiratory rate and volume that persists throughout the acceleration and for some time after the acceleration stops.

In a further investigation Barr[11] (1963) reports the results of respiratory and arterial studies in nine subjects exposed to $+5G_z$ acceleration for 2 minutes with a rate of onset of 1G per 2 seconds. Inspired air was monitored and expired air was collected in Douglas bags for analysis. Blood samples were collected by means of an indwelling radial artery catheter.

During the runs, expired minute volume increased from 8.6 to 20.8 liters per minute and effective alveolar ventilation increased from 4.9 to 9.6 liters per minute. The difference between arterial and end-tidal CO_2 increased by 8.0 mm Hg and was responsible for the major part of the accompanying decrement in end-tidal CO_2 tension. Oxygen uptake increased from a prerun value of 269 to 410 ml per minute, whereas CO_2 elimination increased from 216 to 391 ml per minute, resulting in a change in the respiratory exchange ratio from 0.80 to 0.96. Barr relates the large difference between arterial and end-tidal CO_2 to the result of ventilation of an unperfused portion of the lungs, equivalent to one-third of the total number of alveoli.

Respiration problems will be further discussed in connection with $+G_x$ acceleration.

Electrocardiography

With regard to ECG changes under acceleration, the early workers summarized by Gauer[78] (1950) described changes in the electrical axis, along with some S–T segment and nonspecific T-wave changes. The most definitive study, however, appears to be by Browne and Fitzsimons[27] (1957) who analyzed ECG, respiration, and some vectorcardiograms on 53 subjects in 366 runs under $+3G_z$ to $+5G_z$ for durations of 15 seconds. The subjects were divided into two groups, experienced and inexperienced. Results showed a progressive, and expected, increase in pulse rate with increase in G. At the 3G level, however, the increase in the inexperienced group was significantly greater and was reached in a significantly longer time than with the experienced group. On the other hand, at higher levels of G there was no significant difference between the groups.

The authors argue, with reason, that this

indicates the rise in pulse rate is influenced by an adrenal medullary response, occasioned by apprehension, in addition to the carotid sinus stimulus occasioned by the fall in blood pressure. The argument is strengthened by the fact that the increase begins before the onset of the acceleration, whereas the maximum drop in blood pressure is reached some 4 seconds after onset.

The P–R interval shortens concomitantly with the pulse rate. Gauer [78] (1950) points out that under high G stress and high pulse rate (180/min) the P-wave may not be distinguishable from the S–T complex.

According to Browne and Fitzsimons, the ECG shows no characteristic abnormalities in the conscious subject under acceleration except changes in the electrical axis. This has been confirmed by most other workers. As Gauer [78] (1950) and Franks et al.[71] (1945) point out, however, the reapplication of vagal tone and the return of blood to the right heart that occur with cessation of $+G_z$ acceleration are frequently reflected in bursts of cardiac arrhythmia, bradycardia, marked sinus arrhythmia, extrasystoles, displacement of the pacemaker, and even heart block.

In all subjects of the Browne and Fitzsimons study, except two with horizontal hearts, the axis became more vertical in the frontal and sagittal planes under acceleration, and where rotation occurred it was in an anticlockwise direction. These changes, however, were small compared with similar changes observed with deep inspiration and cannot be regarded as specific for acceleration, although they probably represent elongation of the heart. Equally, no correlation was found between the electrical axis and the anatomical axis as determined from X-ray, in that changes in electrical axis were found without changes in anatomical axis, and vice versa.

The S–T segment and T-wave changes that were observed are considered to be nonspecific except for those found to be progressive after positional effects have been taken into account. In that situation they are considered to be indicative of cardiac strain and a sign of impending unconsciousness. Marukhanyan [123] (1961) noted that displacements of the S–T segment and T-wave changes become most pronounced 10 to 20 seconds before visual disturbance and suggested they might be regarded as a warning of impending cerebral disturbance.

Both indirect and direct vectorcardiography were carried out in some subjects by Browne and Fitzsimons but were found to be of little value in determining changes in anatomical position of the heart.

NEGATIVE ACCELERATION ($-G_z$)

Because the tolerance of man to negative acceleration is very low, exposure to it is avoided as much as possible, and fortunately, it is not frequently encountered in aerospace maneuvers. In consequence, its effects have been much less thoroughly investigated than have those of acceleration in other vectors.

However, the problems that arise are still predominantly hydrostatic in nature. The earliest classical study was that of Jongbloed and Noyons [102] (1933), who showed that exposure of rabbits to $-2\frac{1}{2}G_z$ would cause a bradycardia. By denervating the carotid sinus they demonstrated that this was of carotid sinus origin. Ryan et al.[144] (1950), publishing wartime work on several hundred subjects, showed that, in addition to bradycardia, heart block and prolonged asystole (9 sec) could arise in subjects exposed to accelerations up to $-3G_z$. They further showed that the effects were identical with those that accompany pressure on a sensitive carotid sinus.

It was left for Gamble et al.[75] (1949) to show definitively the extent of the hydrostatic effects. They found that with the onset of acceleration, the arterial and venous pressure rise some 70 to 90 mm as measured in the carotid artery and jugular vein. An adequate arterial-venous (A–V) difference is initially maintained, but with increase in carotid sinus pressure, consequent on the increase in hydrostatic pressure, the resulting vagal stimulation produces bradycardia, decrease in cardiac output, and a secondary fall in arterial pressure while the venous pressure

is still artificially maintained. Thus, the A–V difference approaches zero, and confusion or unconsciousness may arise. Gamble et al. showed that sectioning the vagus of animal subjects prevents the bradycardia and maintains the A–V difference. Their studies also confirmed the occurrence of arrhythmias.

Petechial hemorrhages in the face and conjunctivae are common. Similar damage might be expected in the brain, and in fact, rupture of the posterior communicating branch of the circle of Willis has been demonstrated in goats at $-5G_z$. Rushmer et al.[143] (1947) found that cerebrospinal fluid (CSF) and venous pressure varied simultaneously and correlatively in cats, as measured in the head and neck, and concluded that the CSF pressure at any level reflected the venous pressure at that level. Consequently a change in the intravascular venous pressure would be balanced externally by an increased CSF pressure. They noted, however, that at some time during exposure to negative acceleration a pressure differential, arterial-venous or arterial-CSF, could arise, amounting to a maximum (as measured) of 61 mm Hg.

This differential appeared to Beckman [13] (1949) inadequate to cause the arterial rupture which had been found in goats, and consequently he proceeded to determine whether the A–V difference was of the same order in goats as in cats. Using elaborate and sophisticated techniques he exposed goats to accelerations ranging from $-1.2G_z$ to $-8.9G_z$ and found a close correlation between CSF and venous pressures. Postmortem, the animals showed no indication of cerebral hemorrhage. The average change of venous pressure per G was 26.1 mm Hg, while the change of CSF pressure was 28 mm Hg per G. The greatest A–V difference was found to be 250 mm Hg. This occurred not with the highest arterial pressure, which was found in one animal at $-2G$, but on the occasion of maximum acceleration ($-8.9G$). Equation of an A–V pressure differential with an arterial-CSF pressure differential, as shown above, means that the arterial wall under these circumstances is called upon to support a pressure gradient of 250 mm Hg. Since postexposure A–V differences of 200 mm Hg were observed, this is not considered excessive. Thus, although these figures are greater than those found in cats, they do not appear high enough to cause arterial rupture. However, it must be remembered that no hemorrhage was found in this series, either grossly or histologically. Consequently, while the findings are valid, it may well be that in cases where rupture occurs a preexisting high A–V difference or a defective vessel may be present. This situation remains somewhat academic but unresolved.

As an incidental finding, Beckman shows graphically that the change in systolic pressure per G unit decreases with increasing negative acceleration to an as yet unestablished asymptote. He argues that since the highest systolic pressure was observed at $-2G$, the A–V pressure difference would be greatest at $-2G$ and would decrease above that level. This is not a valid assumption, however, because arterial and venous pressures are subjected to the same hydrostatic pressure and the A–V difference is not governed only by the applied acceleration.

The question of redout demands some consideration. Its existence in operational flying is doubtful, although it has been reported experimentally by Ryan et al.[144] (1950) on the centrifuge. If it actually occurs consistently, and this is debatable, the cause is doubtful. There is no evidence that it is of intraocular or cerebral origin (Christy,[39] 1961). It has been suggested, and Shaw [150] (1948) attributes the suggestion to W. R. Franks, that redout is a distortion of vision caused by looking through the conjunctiva of the lower lid which is pulled upward over the eyeball by the negative acceleration. This is the commonly accepted explanation although it does not appear entirely satisfactory.

Little work has been done on other aspects of negative acceleration; in particular, little is reported on the effects of respiration. Armstrong and Heim [6] (1938) state that there is a slight increase in rate and depth of respiration to a level of about $-3G_z$, above which

the breath is held. The records of Gamble et al.[75] (1949) confirm this.

FORWARD ACCELERATION ($+G_x$)

Whereas the chief physiological limitations in positive acceleration ($+G_z$) arise from the cardiovascular response to increased hydrostatic pressure, the increase in hydrostatic pressure in the forward vector ($+G_x$) is much less because of the shorter distances. In this vector the limiting problems are largely respiratory in character, although some other hydrostatic effects are manifest in the eye and brain if the magnitude is great enough.

Although the early workers, Gauer and Ruff, Bührlen, and others, observed respiratory problems in forward acceleration, most of the definitive work in respiratory dynamics has been carried out in the last few years. Much of this work has been done by the Acceleration Section of the Biomedical Laboratories of Wright Air Development Division and is included in papers such as those by Cherniack et al.[38] (1961), Clark et al.[44] (1959), Watson et al.[175] (1960), Watson and Cherniack [174] (1962), Zechman et al.[197] (1960), Zechman and Taylor [199] (1962), and Zechman and Mueller [198] (1962).

In addition, Hershgold [94] (1960) showed by X-ray studies that during forward accelerations of 6 and $12G_x$ there is elevation of the posterior half of the diaphragm, decrease in the anteroposterior diameter of the thorax, diminution of lung area, and increase in radiolucency in the anterior portion of the lung, which he attributes to decreased perfusion. The heart and trachea are displaced posteriorly. X-ray studies in dogs undertaken by Russian workers showed similar changes (Agadzhanyan and Mansurov,[1] 1962).

Mechanics of Breathing

The respiratory rate increases almost linearly with acceleration. Minute volume increases initially, and then levels at about 8G. Tidal volume, although increasing initially in the fully supine position, decreases with still greater G. Some variations occur according to back angle (fig. 5).

Lung volumes under $+G_x$ are shown in figure 6. Decreases occur in expiratory reserve volume, tidal volume, total lung capacity, and functional residual capacity. No significant change occurs in residual volume.

Vital capacity similarly shows a decrease with increasing acceleration, until at 12G the vital capacity is little more than the tidal volume, indicating a marked decrease in pulmonary reserve. Cherniack et al.[38] (1961) point out that with increased acceleration, both vital capacity and tidal volume would ultimately approach zero, and tolerance without respiratory aids would be the same as breath-holding time.

Barer et al.[8] (1963), in Russia, investigated some aspects of the mechanics of breathing at accelerations up to $+15G_x$. They showed that respiratory rate increased proportionally with acceleration to an average of 29.4 per minute at $+12G_x$. On the other hand, tidal volume increased to $+6G_x$ and then decreased, as follows:

Acceleration, $+G_x$	Tidal volume, cc
1	581
4	637
6	923
8	834
10	788
12	704
15	80 (minimum)

The curve for minute volume was S-shaped with inflection points at 5 and $10G_x$ and an eventual fall-off, while the vital capacity followed a mirror image of the same curve, decreasing where the former increased, up to the point of fall-off.

Thus, a marked and increasing interference occurs in pulmonary ventilation. Figures for maximum breathing capacity, vital capacity, and 0.5-second vital capacity (table 4) show, however, that under accelerations of 3 and $5G_x$, timed vital capacity decreases to a greater degree than does maximum breathing capacity. From this Cherniack [38] (1961) concludes that any defect that occurs

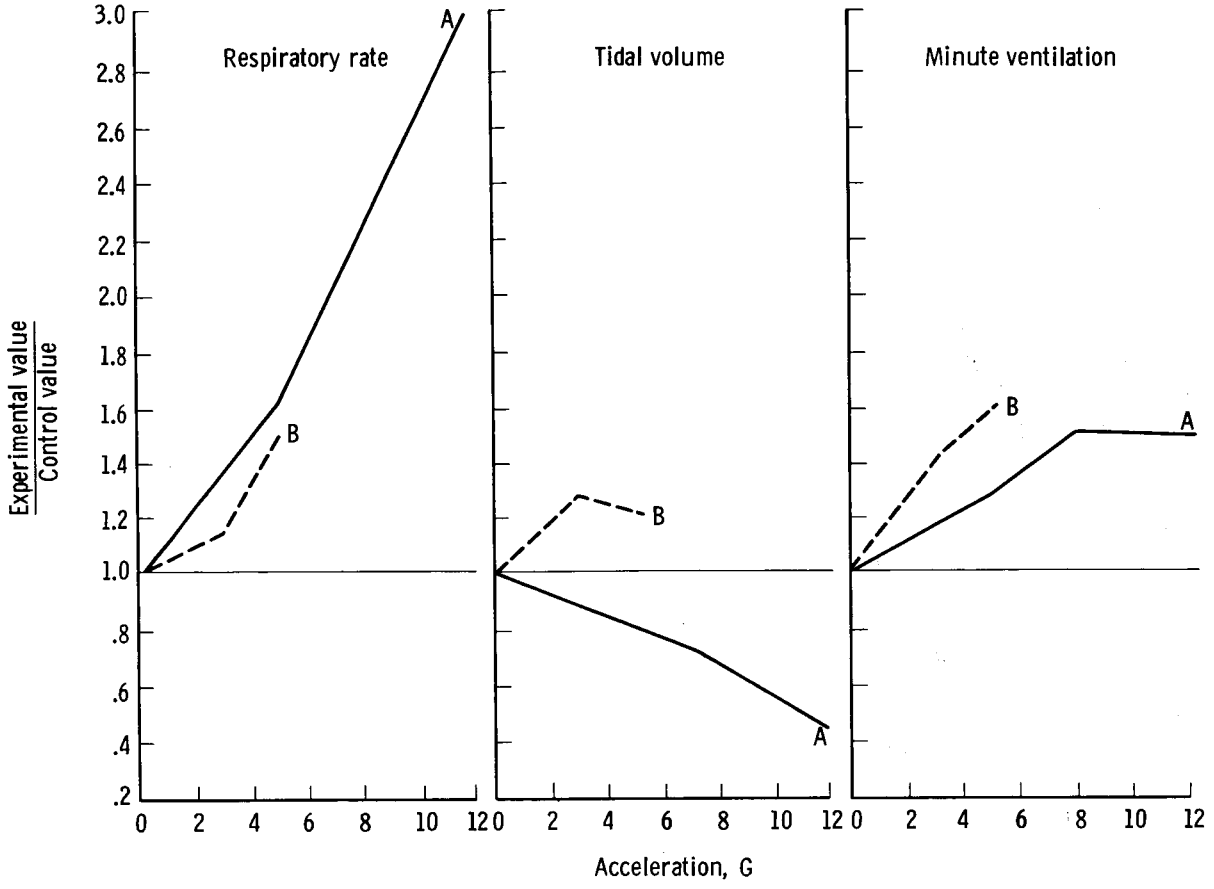


FIGURE 5.—*Effect of back angle on respiration in room air. Curves labeled A represent measurements made on net seat with a 12° back angle, at 5, 8, and 12G. The B curves represent measurements made on a rigid support with a 0° back angle, at 3 and 5G.* (Cherniack et al.,[38] 1961.)

TABLE 4.—*Means and standard deviations for maximum breathing capacity, vital capacity, and 0.5-second vital capacity under forward acceleration.* [Cherniack et al.,[38] 1961.]

Acceleration	Max. breathing capacity, l/min (a)	Vital capacity, cc (b)	0.5-sec vital capacity, % (c)
$+1G_x$	106.3 ± 22.2	4,314 ± 903	52.1 ± 16.7
$+3G_x$	84.5 ± 21.1	2,392 ± 483	61.8 ± 17.7
$+5G_x$	59.2 ± 18.8	1,109 ± 508	76.3 ± 17.9

[a] 12 subjects.
[b] 14 subjects.
[c] 14 subjects.

is restrictive rather than obstructive, the restriction of course being related to the increased pressure on thorax and abdomen.

In an excellent paper, Watson et al.[175] (1960) term this pressure P_G and relate it to the similar force found in negative-pressure breathing, such as occurs when breathing ambient air through a snorkel under water. They showed by constructing pressure-volume loops and static relaxation pressure-volume curves for 1 to $4G_x$ that this force increases linearly with acceleration. Thus, in the situation illustrated in figure 7, the additional resistance at 4G is equivalent to approximately 15 mm Hg, or 5 mm Hg per G, and during acceleration the relaxation pressure at each increment of G is greater than the resting relaxation pressure by the pressure P_G.

Extra work is required, of course, to com-

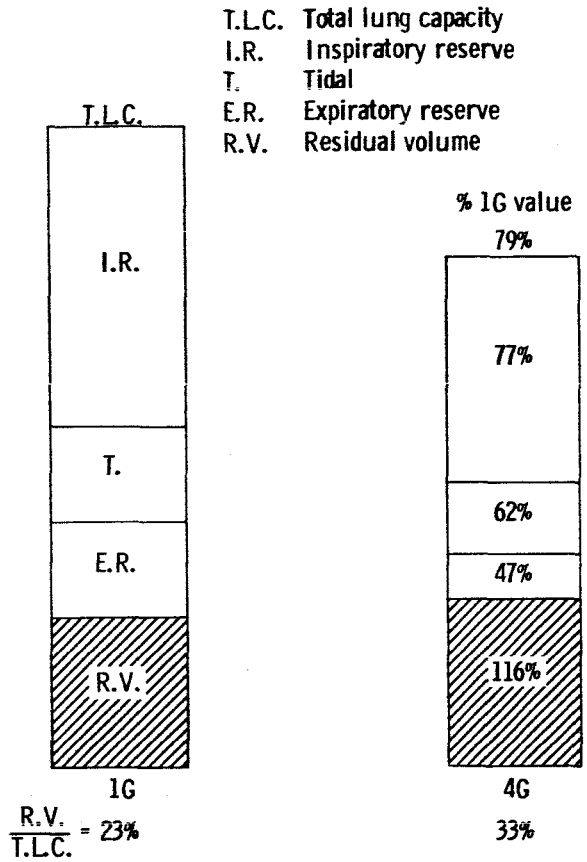


FIGURE 6.—*Changes in lung volumes during forward acceleration.* (Cherniack et al.,[38] 1961.)

bat this increased force. Tables 5 and 6, from Watson's study, show that the total work of breathing nearly doubled from 1 to 4G, with the increase taking place in the elastic work component.

In addition, as will be noted later, hydrostatic changes under $+G_x$ give rise to alterations in the blood distribution in the outer regions of the lungs, that is, posterior in the $+G_x$ position. This might be expected to alter the compliance. Although the static relaxation pressure-volume curves illustrated in figure 7 do not differ in slope and thereby indicate no change in compliance, the authors state that when compliance is measured from the end-expiratory position at each G and over a pressure change of 5 mm Hg, the static total pulmonary compliance becomes progressively smaller with increasing acceleration.

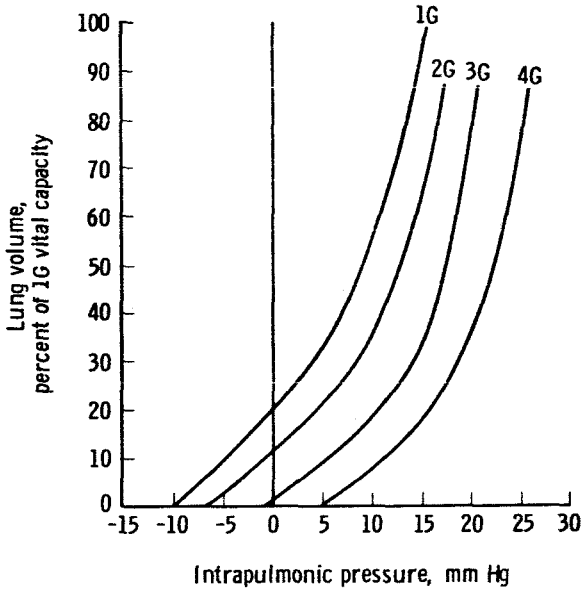


FIGURE 7.—*Static relaxation pressure-volume curves during control (1G) and 2, 3, and 4G forward acceleration. All lung volumes were obtained at 1G.* (Watson et al.[175] 1960.)

The implication is that since the relaxation pressure-volume curves are parallel the decrease must be the result of a fall in the functional residual capacity and pulmonary midposition. Tables 5 and 6 show that the total dynamic compliance also decreases markedly from 1 to 4G. It would be of value to measure the compliance of different lung regions.

It will also be noted from figure 7 that at 4G the relaxation pressure is always positive, even at zero lung volume. Watson et al. suggest that this result is due to pooling of blood or air trapping in the alveoli. However, this is not necessarily so, since the transducer may in fact be reflecting the pressure applied by the acceleration.

The increase in respiratory frequency is marked, and it is linear at least to $+12G_x$. The cause is debatable. Some of the increase is no doubt due to a hypoxic drive and some may be due to chest compression. To investigate this, Zechman and Taylor [199] (1962) exposed six dogs to $6G_x$ for 30 seconds, and also independently to chest compression, measuring frequency of respiration. In each situation a response of increased frequency was

TABLE 5.—*The work of breathing and total dynamic compliance* [a] *at 1G.*
[Watson et al.,[175] 1960.]

Subject	Tidal, cc	P_t, cm H_2O	f	$\dot{V}_E$, l/min	Work of breathing, kg/m/min			Dynamic compliance, cc/cm H_2O
					Elastic	Nonelastic	Total	
A	2,220	27	7	15.5	2.10	0.90	3.00	82
	2,220	27	7	15.5	2.10	.83	2.93	82
	2,220	27	7	15.5	2.10	.93	3.03	82
B	2,070	30	10	20.7	3.07	.62	3.69	70
	2,210	31	10	22.1	3.37	.55	3.92	73
	2,070	30	10	20.7	3.12	.59	3.71	70
C	2,000	29	10	20.0	2.93	1.02	3.95	68
	1,840	30	13	24.0	3.55	1.31	4.86	62
	1,980	29	10	19.8	2.90	1.22	4.12	68
D	1,880	28	13	24.0	3.38	1.01	4.39	68
	1,840	28	13	24.0	3.38	.87	4.25	67
	1,900	28	13	24.7	3.47	1.50	4.97	68
Mean	2,090	29	10	20.5	3.04	.95	3.99	72
Standard error	---	---	---	±0.65	±0.16	±0.09	±0.20	±2

[a] The distensibility of the entire respiratory apparatus, including lungs and chest bellows. P_t=intrapulmonic pressure at the end of the tidal volume; f=respiratory frequency; $\dot{V}_E$=minute ventilation.

TABLE 6.—*The work of breathing and total dynamic compliance* [a] *at 4G.*
[Watson et al.,[175] 1960.]

Subject	Tidal, cc	P_t, cm H_2O	f	$\dot{V}_E$, l/min	Work of breathing, kg/m/min			Dynamic compliance, cc/cm H_2O
					Elastic	Nonelastic	Total	
A	1,210	29	17	20.6	5.20	1.03	6.23	42
	1,280	29	17	21.8	5.55	.84	6.39	44
	1,440	29	17	24.5	6.24	.51	6.75	49
B	840	30	24	20.2	8.23	.51	8.74	28
	950	30	24	22.8	9.30	.26	9.56	32
	950	31	24	22.8	9.30	.39	9.69	31
C	1,120	31	20	22.4	7.06	1.21	8.27	36
	1,120	31	20	22.4	7.06	1.13	8.19	36
	1,000	31	18	18.0	5.65	.94	6.59	33
D	1,020	30	16	16.3	3.87	.77	4.64	34
	1,020	30	16	16.3	3.87	.77	4.64	34
	1,170	30	16	18.7	4.47	.77	5.24	39
Mean	1,093	30	19	20.6	6.32	0.76	7.08	37
Standard error	---	---	---	±0.8	±0.56	±0.08	±0.52	±2

[a] The distensibility of the entire respiratory apparatus, including lungs and chest bellows. P_t=intrapulmonic pressure at the end of the tidal volume; f=respiratory frequency; $\dot{V}_E$=minute ventilation.

elicited. Section of the vagi abolished the response to compression but not to acceleration. Denervation of the carotid bodies alone did not abolish the response to acceleration, but section of the vagi and denervation of the carotids prevented the response in three out of four dogs. The authors argue that this indicates the existence of a hypoxic drive mediated by chemoreceptors in the carotid bodies, pointing out that the aortic body would continue to act as a chemoreceptor while the vagi were intact. This is not entirely convincing and requires further investigation.

Effects of Pressure Breathing

In a followup of their observation that the effect of forward acceleration is similar to

that found in negative-pressure breathing, Watson and Cherniack [174] (1962) investigated the effects of positive-pressure breathing on respiratory mechanics during acceleration of 12 normal subjects at 4, 6, and 8G_x.

Using the technique of electrical integration of a pneumotachygraph record to derive volumes, they compared vital capacity, inspiratory reserve, tidal air, and expiratory reserve under conditions of oxygen breathing at ambient and applied positive pressure. The results, shown in tables 7 and 8, indicate that positive-pressure breathing (PPB) diminishes the changes in lung volumes resulting from forward acceleration.

On the basis that forward acceleration increases intrapulmonic pressure by 5 mm Hg per 1G increment in acceleration for constant lung volume, as was noted in a previously mentioned study (Watson et al.[75] 1960), the theoretical applied pressures would be 25 and 35 mm Hg at 6 and 8G, respectively. This was, however, found to be in excess of the actual tolerable pressure gradient, namely, 2½ to 3 mm Hg per G. The explanation is not clear, although it is possible that the 5 mm may represent a peak pressure, whereas the 3 mm Hg represents a mean.

A tolerance study with and without positive-pressure breathing was also undertaken. There was a 67% increase in tolerable duration of exposure with the pressure breathing at 10G_x, to a maximum in one case of 5½ minutes.

It will be noted, of course, that this study was carried out with oxygen and not atmospheric air. Considering that atelectasis occurs more readily when oxygen breathing is combined with acceleration (Ernsting,[66] 1960), it would be advisable to carry out studies of this nature with pressurized air, with oxygen at reduced pressures, and also with atmospheres currently contemplated for space cabins. A picture could then be obtained of the situation uncomplicated by excess oxygen.

TABLE 7.—*Mean lung volume changes during increasing forward acceleration ± standard error.*[a] [Watson and Cherniack,[174] 1962.]

Acceleration (b)	Vital capacity		Tidal		Inspiratory reserve		Expiratory reserve	
	cc	% of 1G value	cc	% of 1G value	cc	% of 1G value	cc	% of 1G value
1G	3,872 ± 343		781 ± 107		2,333 ± 191		76 ± 135	
4G	2,629 ± 460	68	484 ± 71	62	1,785 ± 310	77	360 ± 94	47
6G	1,867 ± 252	48	452 ± 17	58	968 ± 241	41	447 ± 130	59
8G	1,216 ± 160	31	378 ± 31	48	582 ± 171	25	274 ± 76	36
10G	570 ± 59	14						

[a] Each figure is the mean for four or five subjects.
[b] 1G–8G studies done with an A–13–A mask and 12° back angle; 10G studies done with an MA_2 full-pressure helmet and 0° back angle.

TABLE 8.—*Effect of positive-pressure breathing on mean lung volumes during increasing forward acceleration ± standard error.*[a] [Watson and Cherniack,[174] 1962.]

Accel. (b)	With PPB mm Hg	Vital Capacity		Tidal		Inspiratory reserve		Expiratory reserve	
		cc	% of 1G value	cc	% of 1G value	cc	% of 1G value	cc	% of 1G value
1G	0	3,875 ± 343		781 ± 107		2,333 ± 171		761 ± 135	
4G	12	3,642 ± 535	94	485 ± 56	62	2,089 ± 343	90	1,180 ± 121	155
6G	16	3,121 ± 565	81	435 ± 45	56	1,681 ± 397	72	1,005 ± 146	132
8G	20	2,391 ± 715	62	510 ± 122	65	947 ± 407	41	938 ± 271	123

[a] Each figure is the mean for four or five subjects.
[b] 1G–8G studies done with an A–13–A mask and 12° back angle; 10G studies done with an MA_2 full-pressure helmet and 0° back angle.

Pulmonary Gas Exchange

There is little doubt that changes occur in pulmonary gas exchange during G_x acceleration. This matter has been investigated by the Wright Air Development group. Zechman et al.[197] (1960) made estimates of oxygen consumption in subjects exposed to 5, 8, 10, and 12G_x for 1 to 2 minutes. Using a spirometer technique, they observed a marked shift in the expiratory midposition on the tracing during exposure to acceleration. Estimation of the oxygen cost of acceleration was based on the difference between the preacceleration control slope and the postacceleration slope. This indicated the total additional oxygen consumed during onset, acceleration, and offset.

The results are shown in table 9 and could be interpreted as indicating a progressive increment in O_2 uptake with increasing G. The standard deviations, however, are so large that the numbers are less useful than they might have been. Increase in O_2 uptake under $+G_x$ acceleration was also observed by Smedal et al.[155] (1963).

In another part of this series, Zechman et al.[197] (1960) measured the nitrogen washout from the lungs during a 30-second period of acceleration which was preceded by 30 seconds of oxygen breathing (*i.e.*, a total of 1 minute's acceleration), and compared it with a similar period at 1G. The nitrogen eliminated during a given period of oxygen breathing depends on the adequacy of the alveolar ventilation, the stability of the functional residual capacity (Agostini et al.[2] 1959), and the volume of nitrogen excreted from the blood. The excreted nitrogen in the time period is alleged to be negligible, the functional residual capacity was not increased, and the nitrogen eliminated was the same for acceleration and control (table 10). Zechman et al. argue that these results indicate no gross change in alveolar ventilation.

This argument, however, must be predicated on steady-state conditions. Steady state would not have been reached in 30 seconds, and, furthermore, the functional residual capacity actually decreases during forward acceleration (Watson et al.[175] 1960) and no doubt varied dynamically during the development of acceleration. Also, it is unreasonable to assume that no significant amount of nitrogen will be eliminated from the bloodstream during the 30-second period. The argument must therefore be regarded as invalid.

In this connection Smedal et al.[155] (1963) made the interesting observation that immediately after 2 minutes' exposure to $+4G_x$ or $+6G_x$ there was a small but rapid rise in their closed-circuit nitrogen content, equal to 100 to 150 ml of extra nitrogen. They attributed this either to return to the pulmonary circulation of previously pooled venous blood in equilibrium with air where it would be exposed to alveoli washed out with oxygen, or alternatively to the return to a ventilated state of alveoli occluded by thoracic compression. Each explanation seems reasonable and either or both could play a part.

TABLE 9.—*Additional oxygen consumption for 1 minute of forward acceleration (cc/min STPD).* [Zechman et al.,[197] 1960.]

Subject	5G		8G		10G		12G	
	Control	Extra	Control	Extra	Control	Extra	Control	Extra
A	324	0	312	174	293	266	330	461
B	311	0			286	594		
C			286	459				
D	271	0			312	124	294	368
E							294	1,321
F			352	270	293	569		
G	288	0	311	18				
H	238	55	253	208	239	293		
I			226	0	221	18		
J	292	220						
Mean ± S.D.	287 ± 32	46 ± 78	290 ± 45	188 ± 170	274 ± 34	310 ± 228	306	716

TABLE 10.—*Effect of forward acceleration on respiratory frequency, tidal volume, minute volume, and nitrogen elimination.* [Zechman et al.,[197] 1960.]

	Frequency per min	Tidal vol., cc (ATPS)	Min. vol., l/min (ATPS)	N_2 elim., l/30 sec
Control	13.8 ± 4.0	580 ± 125	8.0 ± 1.2	1.33 ± 0.19
5G	22.2 ± 8.4	491 ± 179	10.9 ± 2.6	1.60 ± 0.42
Control	13.4 ± 4.2	635 ± 204	8.5 ± 2.6	1.55 ± 0.34
8G	29.6 ± 14.2	411 ± 140	12.2 ± 2.0	1.53 ± 0.22
Control	14.4 ± 4.4	590 ± 243	8.5 ± 1.8	1.51 ± 0.30
12G	39.2 ± 9.2	318 ± 208	12.5 ± 5.6	1.40 ± 0.42

TABLE 11.—*Minute volume of ventilation, oxygen uptake, carbon dioxide excretion, and respiratory exchange ratio at 6G and 8G forward acceleration.*[a] [Steiner et al.,[165] 1961.]

	$\dot{V}_I$ l/min (ATPS)	$\dot{V}_{O_2}$, ml/min (STPD)	$\dot{V}_{CO_2}$, ml/min (STPD)	R
Prerun	6.94 ± 0.8	275 ± 24	231 ± 31	0.83 ± 0.12
6G, 3 min	[b]11.80 ± 2.8	259 ± 90	[b]293 ± 71	[b]1.17 ± 0.39
1–3 min postrun	[b]11.17 ± 2.1	[b]403 ± 95	[b]328 ± 52	0.81 ± 0.16
3–6 min postrun	8.93 ± 0.46	304 ± 80	254 ± 69	0.86 ± 0.04
Prerun	7.63 ± 1.25	276 ± 50	228 ± 50	0.81 ± 0.16
8G, 3 min	[b]11.07 ± 3.8	[b]205 ± 60	259 ± 92	[b]1.25 ± 0.39
1–3 min postrun	[b]15.06 ± 4.9	[b]477 ± 100	[b]396 ± 130	0.80 ± 0.14
3–6 min postrun	9.57 ± 3.74	[b]326 ± 61	254 ± 97	0.82 ± 0.13

[a] Values are means ± standard deviation of means.
[b] $P < 0.05$ (compared to prerun controls).

Steiner et al.[165] (1961) examined further the question of oxygen consumption in a paper that has received some attention. They exposed seven experienced subjects to forward accelerations of $+6G_x$ and $+8G_x$ at 0.5G/sec rate of onset, maintaining a 3-minute plateau. The total volume of expired gas for the 3-minute plateau was collected, as were two consecutive 3-minute postrun collections. Gas volumes, oxygen partial pressure, and carbon dioxide percentages were determined.

Table 11 shows the results, which the authors claim indicate a reduced oxygen uptake during acceleration followed by an increased uptake postacceleration, greater than required to repay the oxygen debt incurred. They go on to state that "the additional O_2 requirement for the added metabolic work during acceleration is approximately 141 ml/min at 6G and 147 ml/min at 8G."

Several things may be noted about the numbers in the table and statement. The standard deviations in the table for minute volume of oxygen uptake and CO_2 excretion are remarkably high—so much so that at the 6G level there is no significant difference in oxygen uptake from control values, nor between the second postrun and control levels. At the 8G level the difference is a little more striking for oxygen, but not significant for CO_2. At best one might say there is a trend toward decrease in oxygen uptake during acceleration, followed by an increase immediately postacceleration, and for increased CO_2 excretion.

Again, if one examines the statement quoted for the "additional oxygen requirement," namely, 141 ml/min at 6G and 147 ml/min at 8G, one may confirm the former figure by simple calculation from the tables. It would appear, however, that the latter figure should read 180 ml/min instead of 147 ml/min.

As to the significance of the results, the authors note that steady state has not been

established, but accept the figures as though they were representative of steady-state conditions and show a reduction in O_2 consumption and an increase in CO_2 excretion during acceleration in the face of an adequate oxygen supply, followed by a large increase in O_2 consumption postacceleration.

Assuming that the facts are valid, they would reflect a reduction in oxygen reserves. A reduction in alveolar oxygen, or oxygen in arterial or venous blood, tissue fluid, or myoglobin, will result in decreased oxygen uptake during the period of time over which this reduction is taking place. Such a reduction could arise from reduced cardiac output, hypoventilation, disturbed diffusion capacity, or disturbed ventilation-perfusion relationships. Reduction in cardiac output, however, is probably of relatively little significance since it has been shown to fall only 18% at 3G (Lindberg et al.[116] 1960). Equally, alveolar ventilation is normal or increased at these levels of acceleration. There is no doubt that the diffusion capacity is markedly affected, as will be discussed, but probably the major factor is altered ventilation-perfusion relationships.

At this point it is well to note that Glaister [85] (1963) carried out a somewhat similar experiment at $+2G_z$ to $+3G_z$ for ½ to 5 minutes. Although he presents his data in averages, without standard deviations, he too observed that the oxygen uptake during acceleration failed to increase, or even fell, and there was a postacceleration oxygen debt of as much as 500 to 600 ml. He also carried out single-breath carbon dioxide tests on several subjects at $+3G_z$, comparing the results with similar tests at $+1G_z$. Normally the CO_2 concentration in the expired air rises rapidly as the anatomical dead space is washed out and then remains at a plateau level, or even rises slightly. At $+3G_z$ the plateau was observed to be lower and to fall off slightly after the expiration of some 1500 ml of air. This fall-off could also be demonstrated at $+18G_z$.

Glaister refers to a comparable falling plateau that occurs when the blood supply leading to one lobe of the lung of a dog is occluded and when the bronchus leading to the same lobe is constricted, and observes that the operating mechanism is late emptying of an underperfused area of the lung. He relates the findings under acceleration to local differences in ventilation and perfusion, such that during $+G_z$ acceleration the lower lobes of the lung are overperfused and underventilated while the upper lobes are overventilated and underperfused. Inequalities of this nature would have the same effect on arterial blood as pulmonary shunt, with a fall in oxygen tension and a rise in CO_2 tension. Similar conclusions with respect to $+G_x$ acceleration were reached by Steiner et al.[165] (1961), with more devious reasoning, on the basis of their previously noted findings. Wood et al.[195] (1961), as will be discussed, confirmed by other methods that pulmonary shunting does take place under conditions of forward acceleration.

Glaister [85] (1963) also notes that the fall in oxygen reserves that results from lowered oxygen tension would explain the time course and magnitude of the changes seen in oxygen uptake during acceleration but is too small to account for the large increase in oxygen uptake following acceleration. He argues that to provide an oxygen debt of 500 ml the arterial oxygen saturation would have to fall to about 50% during acceleration, which he considers unusually low. Accordingly he postulates that the bulk of the oxygen debt represents an increase in metabolism that occurs during the preceding acceleration and appears as a debt due to the building up of products of anaerobic metabolism. Since the reasons for such anaerobic metabolism are as yet unclear, further investigation of this hypothesis is necessary.

In another field, following up the observation of the relationship between the respiratory effects of forward acceleration and negative-pressure breathing (NPB), Zechman and Mueller [198] (1962) undertook a comparison study, measuring pulmonary gas exchange and diffusion capacity in nine human subjects during NPB (-15 mm Hg) and forward acceleration ($+4G_x$). They observed

an increased pulmonary ventilation of approximately 40% in each case, although with NPB the increase was obtained by an increased tidal volume whereas with forward acceleration the increase was due to increased frequency.

Oxygen uptake increased with negative-pressure breathing and was unchanged or slightly decreased with forward acceleration. Figures are detailed in table 12.

The major difference occurred in diffusion capacity (Dco). This was evaluated with a carbon monoxide technique, and although the authors claim that steady state was reached under acceleration it is unfortunate that they do not state the duration of the acceleration. It is implied that the duration was 6 minutes.

The Dco under negative-pressure breathing was unchanged from control values, while with acceleration it dropped from 21.1 ml/min/mm Hg to 12.1. The actual significance of this decrease in diffusion capacity is not yet clear. How much of it is due to the development of pulmonary edema and how much reflects a decrease in the area of functional alveoli in contact with functional capillaries remains to be examined. It is obvious that direct measurement of regional blood flow and ventilation under acceleration is required.

Intrathoracic Pressures

Theoretically, with the increased hydrostatic pressures produced by acceleration in the $+G_x$ vector, pressures in the pulmonary circulation should alter as indicated in figure 8, or in fact be even greater, since the atrial

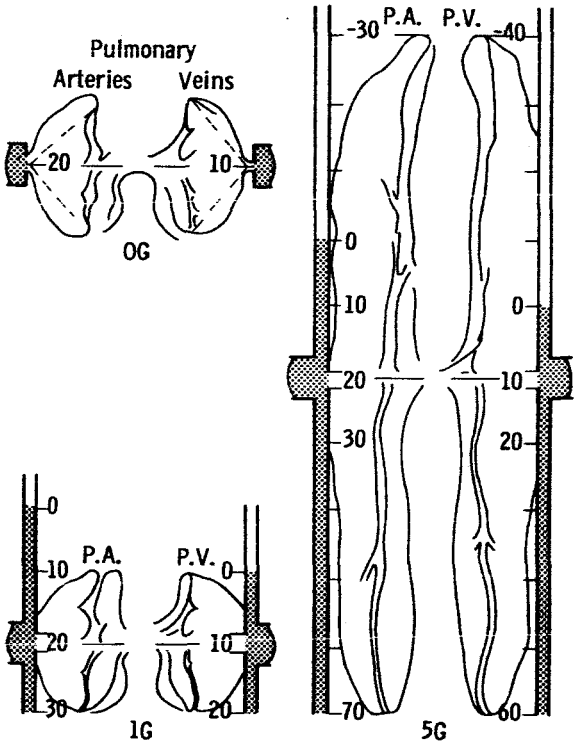


FIGURE 8.—*Diagram of hydrostatic effects of forward ($+G_x$) acceleration on pulmonary circulation. Numbers indicate pressure in cm H_2O; reference 0 is atmospheric pressure at midchest level; dorsal-ventral dimension of lungs is 20 cm.* (Lindberg and Wood,[117] 1963.)

TABLE 12.—*Respiratory effects of negative-pressure breathing and forward acceleration.*[a] [Zechman and Muller,[198] 1962.]

	Control	NPB (− 15 mm Hg)	Control	Forward accel., 4G
$\dot{V}_E$, l/min, BTPS	6.45 ± 0.94	9.13 ± 3.08	6.20 ± 0.92	8.57 ± 1.85
V_T, ml, BTPS	628 ± 144	821 ± 213	709 ± 202	615 ± 138
f, cycles/min	10.7 ± 2.7	11.6 ± 4.0	9.4 ± 3.2	14.5 ± 4.1
P_AO_2, mm Hg	102 ± 3	110 ± 8	101 ± 4	112 ± 5
P_ACO_2, mm Hg	39 ± 2	33 ± 4	38 ± 2	32 ± 4
$\dot{V}_{CO_2}$, ml/min, STPD	215 ± 30	258 ± 51	206 ± 22	230 ± 23
$\dot{V}_{O_2}$, ml/min, STPD	261 ± 29	293 ± 30	246 ± 22	238 ± 29
R	0.82 ± 0.04	0.88 ± 0.12	0.84 ± 0.07	0.98 ± 0.12
Dco, ml/min/mm Hg	20.4 ± 4.1	21.7 ± 6.2	21.1 ± 4.0	12.1 ± 2.7

[a] Average values ± standard deviation for the same nine subjects studied in each condition.

pressures are actually increased under $+G_x$ acceleration, as was shown by Lindberg et al.[115] in 1962, and do not remain unchanged as indicated in the figure. These increased pressures in the dependent regions should produce pulmonary edema, and, in addition, there should be a tendency for an air shunt to develop anteriorly and a blood shunt posteriorly. Hershgold's radiography studies also suggest this.

A diagrammatic representation of these changes is given in figure 9, from a comprehensive paper by Wood et al.[193] (1963) dealing with these matters. They point out that for a lung with a dorsoventral diameter of 20 cm, with contents of an average specific gravity of 0.5, the intrapleural pressure due to the elastic properties of the lung is about -7 cm H_2O in the weightless state. At $1G_x$, because of hydrostatic effects, the ventral intrapleural pressure would be about -12 cm H_2O and the dorsal about -2 cm H_2O. At $+5G_x$ the dorsal pressure would be expected to become about 18 cm H_2O and the ventral -32 cm H_2O, with corresponding changes in the arterial-venous pressures.

Wood points out that at 6G negative pressures in the ventral region can begin to approach the threshold for rupture of pulmonary parenchyma. As illustration, he quotes an occurrence in his laboratory where an acute incapacitating mediastinal emphysema occurred in a healthy subject during exposure to $+5.5G_x$ while he was performing maximal voluntary hyperventilation in an unsuccessful effort to prevent a decrease in arterial oxygen saturation. It is understood that similar results have been found in animals by the Russians.

In the absence of voluntary hyperventilation, however, it is unlikely that this will be an operational problem. Evidence for the occurrence of pulmonary edema is only indirect in man. It includes a demonstrated fall in right and left atrial pressures during continuance of acceleration after the initial rise (fig. 10) which Wood et al. attribute to a loss of plasma volume amounting to nearly 400 ml, and it also includes the occurrence of hemoconcentration as indicated by an increase in the blood optical density at 800mμ in dogs accelerated at $+6G_x$ (Wood,[192] 1963).

Evidence for the change in intrapleural pressure is more direct. Wood [192] (1963) describes a technique of percutaneous intrapleural catheterization in dogs that allows direct measurement of pressure, which can thereafter be referred to the midchest position. An average dorsoventral gradient in the $1G_x$ position was found to be 0.5 cm H_2O

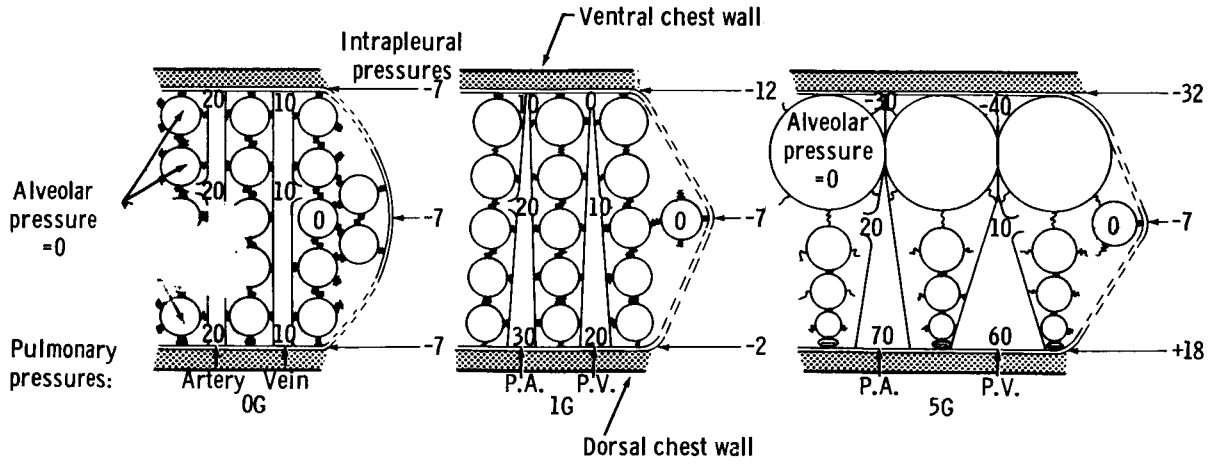


FIGURE 9.—*Diagram of effects of forward ($+G_x$) acceleration on intrathoracic pressures (dorsal-ventral dimension of lung is 20 cm.). Numerals indicate pressures as cm H_2O, and zero reference level is atmospheric pressure at midthoracic coronal plane.* (Wood et al.,[193] 1963.)

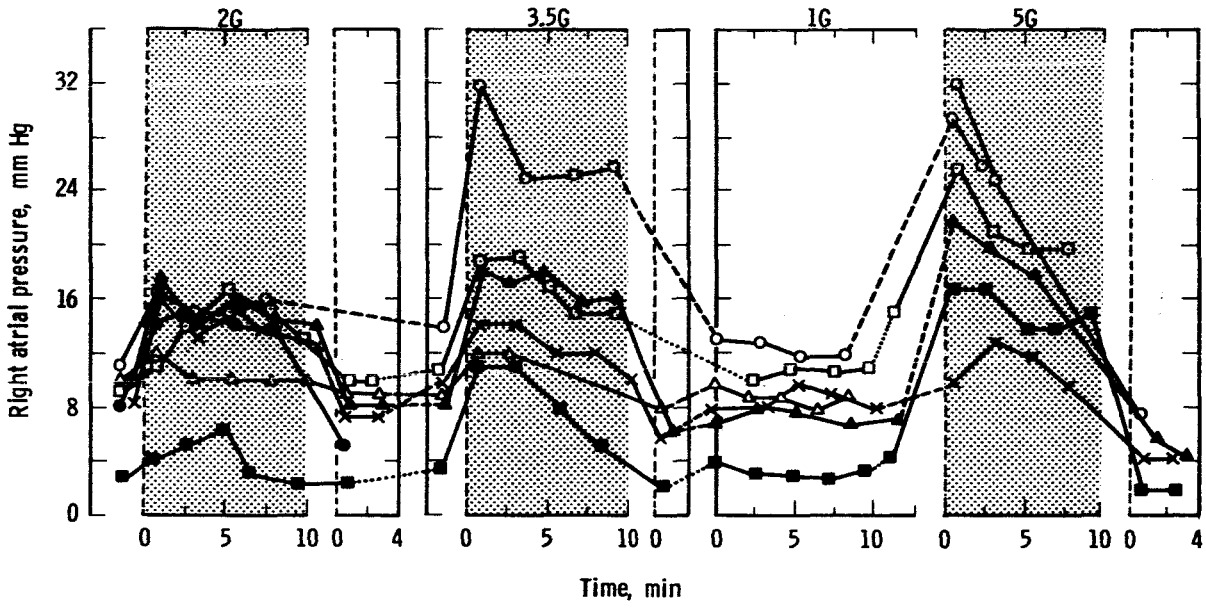


FIGURE 10.—*Effect of forward acceleration on right atrial pressure in six healthy subjects.* (Wood et al.[193] 1963.)

per cm of distance between catheter tips. The gradients for $+6G_x$ acceleration are shown in table 13.

TABLE 13.—*Mean end-expiratory intrapleural pressures at dorsal and ventral sites in thorax of dogs.* [Wood,[192] 1963.]

Acceleration and position		Intrapleural pressures [a]		
		Ventral	Dorsal	Difference, dorsal-ventral (b)
Control (1G)		−11	−4	7
6G	15° head-up	−44	7	51
	Horizontal	−31	17	48
	15° head-down	−25	20	45

[a] Pressures are expressed in cm H_2O referred to ambient atmospheric pressure and are shown as average values from four dogs in supine position.

[b] Average vertical distance between recording sites, 12 cm.

Arterial Oxygen Saturation

Figure 11 illustrates other results of these hydrostatic effects, namely, the development of an arteriovenous shunt and a fall in blood oxygen saturation. It will be incidentally noted that the breathing of 99.6% oxygen delays the development and reduces the magnitude of the desaturation, but does not abolish it.

This, of course, has also been observed in man. Wood et al. in figure 12 show the average and range of changes in arterial oxygen saturation in four healthy men at 2.1, 3.7, and $5.4G_x$ breathing air, while Steiner and Mueller [163] (1961) show in table 14 the changes that occur when breathing air at 6 and $8G_x$ and oxygen at $8G_x$. Assuming a constant cardiac output of 6 liters per minute and using the data for oxygen consumption discussed in connection with table 11, Steiner and Mueller calculated the physiological shunt for 6G and 8G in air and 8G breathing 100% oxygen. They found a 40% shunt at 6G and 60% at 8G in air, and 40% at 8G breathing oxygen. Since the oxygen consumption figures are suspect, however, these findings cannot be wholeheartedly accepted.

An interesting adjunct to this study was the postmortem examination of a lung from a dog sacrificed immediately after acceleration at $14G_x$ for 10 minutes. The anterior portion showed a marked overexpansion of

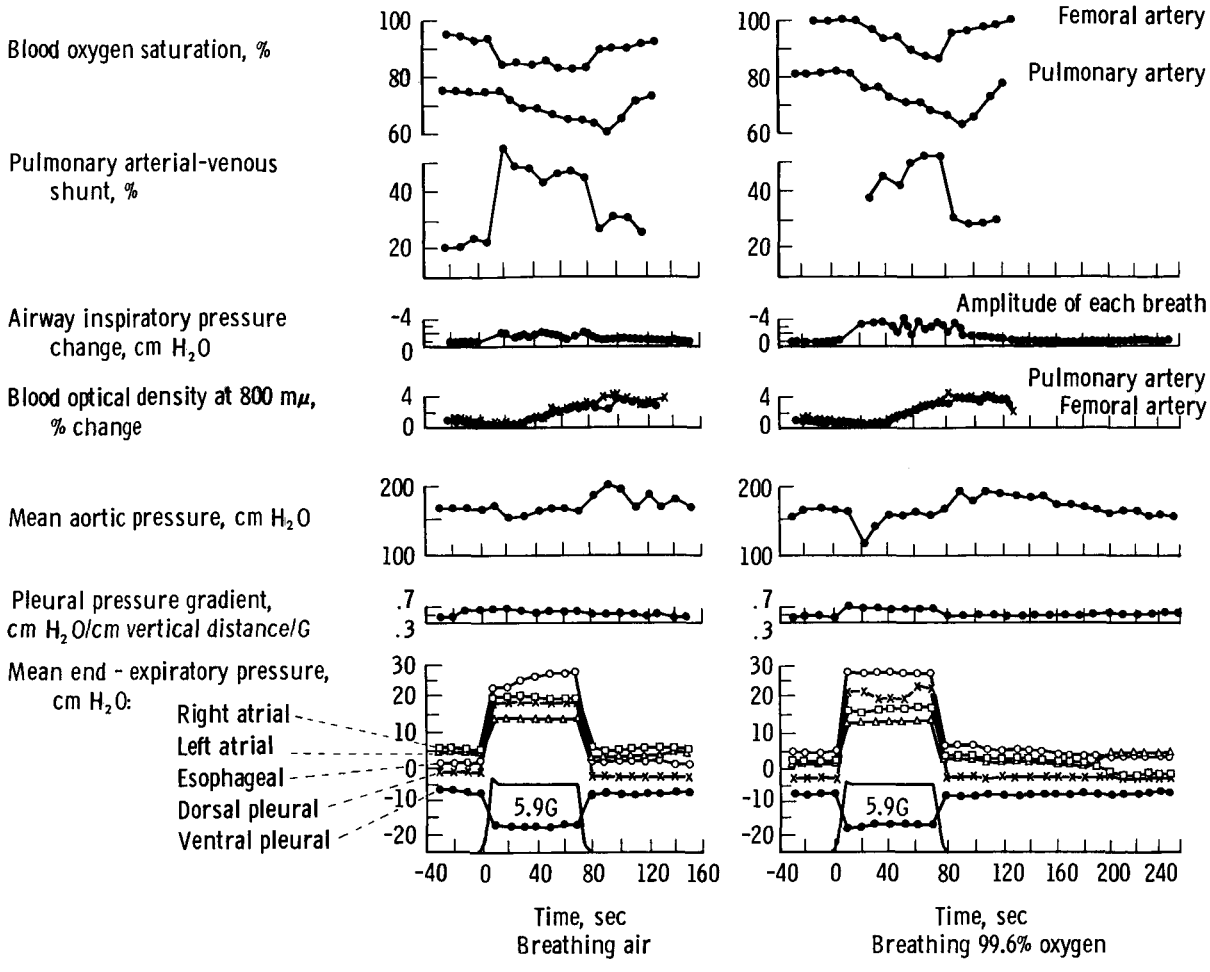


FIGURE 11.—*Changes in simultaneously recorded physiologic variables during exposure of an anesthetized (morphinepentobarbital) 20-kg dog to forward accelerations of 5.9G while breathing air and oxygen at ambient pressure (735 mm Hg).* (Wood et al.,[192] 1963.)

the alveoli and a striking absence of red cells. The middle region showed a relatively normal parenchyma with normal distribution of blood and air. In the dorsal region there were large areas totally devoid of patent alveoli, massive atelectasis, and an increase in blood volume. Surprisingly, there was no evidence of edema. Nevertheless, ample reason exists for reduced diffusion capacity and arterial saturation. Similar postmortem findings in animals were noted by Wood et al.[192] (1963).

Kiselev [104] (1962) investigated changes in the pulmonary circulation of dogs under $+3G_x$, $+6G_x$, and $+9G_x$. Although there is no indication in the available literature of his technique, he measured right ventricular pressure, rate of pulmonary blood flow, arterial oxygen concentration (site unstated), respiration rate, and pulse rate. A direct relationship was found between pulmonary blood flow and oxygen concentration, with a reduction in the latter being observed after 1 to 1½ minutes of acceleration. Kiselev concluded that acceleration leads to marked changes in the hemodynamics of the pulmonary circulation.

In a more recent study undertaken on behalf of NASA, Alexander et al.[3] (1964) measured arterial oxygen saturation in a group of 25 pilots exposed on the Johnsville

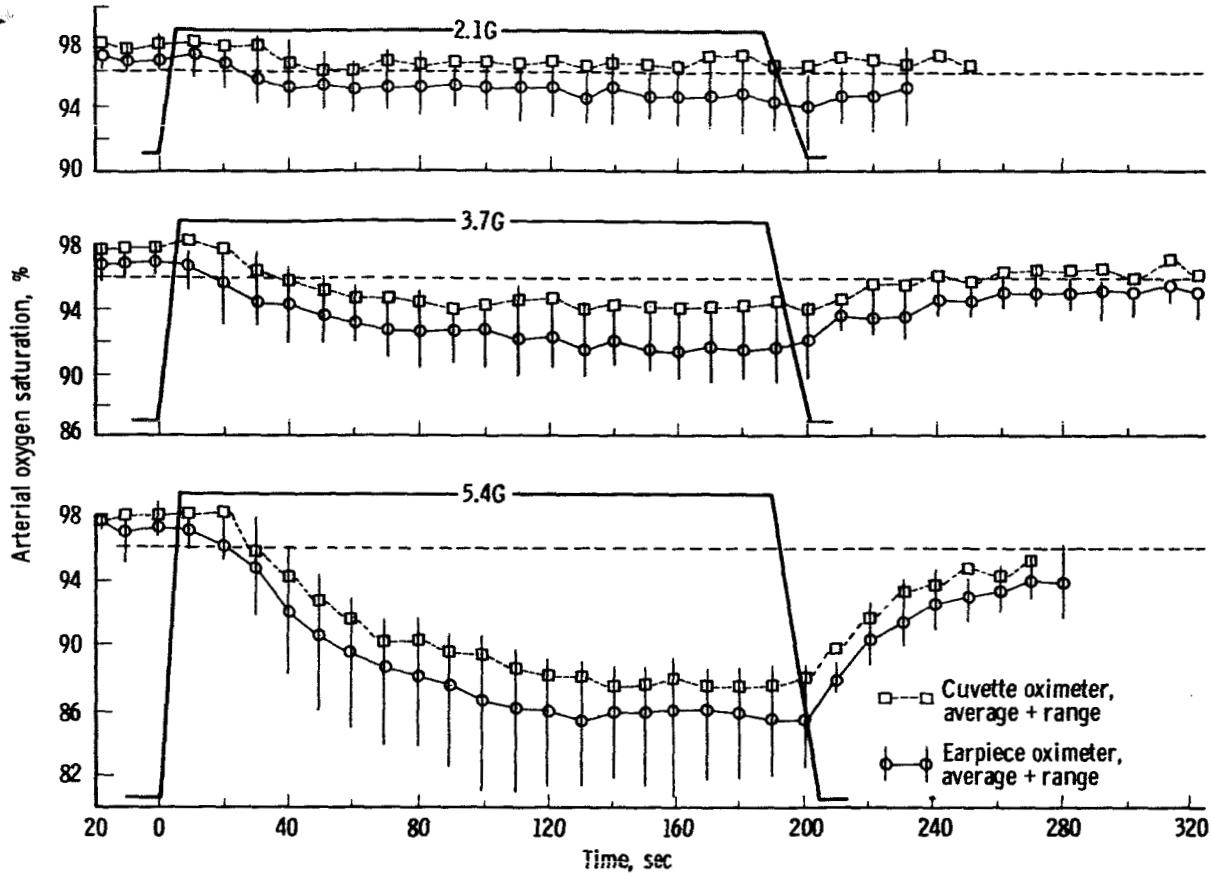


FIGURE 12.—*Average and range of changes in arterial oxygen saturation of four healthy men, recorded by cuvette and ear oximeters during 3 minutes at 2.1, 3.7, and 5.4G, breathing air.* (Wood et al.,[193] 1963.)

centrifuge to trapezoidal $+G_x$ accelerations representative of Apollo launch, reentry, and abort profiles. Using a rigid experimental protocol, they measured saturation in some of the subjects, in the Apollo couch configuration, by both ear oximetry and arterial cuvette. Since the two methods yielded comparable results, the saturation of the rest of the subjects was measured by ear oximetry alone. Other data obtained included ECG, respiratory rate, and radial arterial blood pressure. The last was measured by means of the Mercury automatic system, but unfortunately was unreliable. Minute volume of respiration was determined by integration of the respiratory rate curves.

The saturations obtained while breathing ambient air through a demand regulator and mask system under accelerations of 4, 6, 8, and $10G_x$ of sufficient duration to dissipate a change in velocity of 37,000 ft/sec are illustrated in figure 13. Each curve represents the mean of over 100 exposures for 25 subjects. Standard deviations are not quoted but were of the order of $\pm 5\%$ at $10G_x$.

It will be noted that after an initial rise, probably due to hyperventilation, there is a rapid and almost linear fall (except at the $4G_x$ level) to a minimum of about 67% at $10G_x$. This minimum is then maintained at least as long as the acceleration, in this situation, is applied. On cessation of acceleration there is a rapid climb to about 85% during the first 30 seconds followed by a prolonged recovery over the next 2½ minutes. The irregularity of the $4G_x$ curve is no doubt related to dynamic adjustments of the atelectatic process taking place under conditions

TABLE 14.—*Arterial saturation, whole blood CO_2 content, pH, P_{CO_2}, and whole blood buffer base.*[a] [Steiner and Mueller [163] 1961.]

	O_2 content, vol. %	O_2 capacity, vol. %	% saturation	While blood CO_2 content, vol. %	pH	P_{CO_2}, mm Hg
Air, 5 subjects						
Control	18.98 ± 1.18	19.67 ± 1.3	96.6 ± 3.4	46.1 ± 2.2	7.53 ± .14	32 ± 07.5
6G	[b]16.78 ± 1.09	19.53 ± 1.4	[b]83.7 ± 4.5	[b]41.1 ± 1.5	[b]7.41 ± .12	[b]39 ± 08.9
Recovery, 3 min	18.31 ± 1.27	19.81 ± 1.6	92.4 ± 1.7	44.7 ± 2.9	7.50 ± .16	33 ± 11.0
Recovery, 5 min	18.91 ± 1.27	19.88 ± 1.5	94.7 ± 0.9	44.4 ± 2.5	7.52 ± .11	32 ± 06.2
Air, 6 subjects						
Control	19.15 ± 2.28	19.67 ± 2.9	97.5 ± 3.7	45.3 ± 3.5	7.49 ± .06	33 ± 05.6
8G	[b]13.93 ± 1.90	18.56 ± 2.7	[b]75.5 ± 7.7	[b]39.7 ± 2.1	[b]7.40 ± .10	[b]35 ± 07.3
Recovery, 3 min	[b]17.60 ± 2.02	19.12 ± 1.8	[b]92.3 ± 6.8	[b]42.1 ± 3.6	[b]7.47 ± .05	33 ± 06.4
Recovery, 5 min	18.61 ± 2.19	19.12 ± 1.8	97.2 ± 3.9	43.2 ± 3.3	7.48 ± .05	33 ± 05.8
100% O_2, 8 subjects						
Control	19.71 ± 1.55	19.95 ± 1.6	98.8 ± 1.9	45.2 ± 3.1	7.48 ± .06	35 ± 04.6
8G	[b]16.93 ± 2.30	19.67 ± 1.2	[b]86.2 ± 9.4	44.7 ± 2.3	[b]7.41 ± .06	[b]40 ± 05.2
Recovery, 3 min	19.65 ± 1.48	19.80 ± 1.6	99.0 ± 1.8	[b]43.7 ± 2.8	[b]7.44 ± .05	37 ± 04.0
Recovery, 5 min	19.71 ± 1.55	19.77 ± 1.6	99.8 ± 1.9	44.5 ± 2.5	7.47 ± .04	36 ± 03.7

[a] At end of 3 min during forward acceleration at 6G and 8G breathing room air and breathing 100% oxygen. Values are means ± standard deviation of the means.

[b] Significantly different from control ($P < 0.05$).

insufficient to produce a rapid and consistent atelectasis. Even at $4G_x$, however, saturation falls to a little over 85%.

Figure 14 illustrates the findings for subjects breathing 100% oxygen at 5 psi in otherwise similar circumstances. It will be noted that the desaturation is less (72% at $10G_x$) and the rate of desaturation is slower. In addition the final spread is less, and again the $4G_x$ curve is variable. Very marked, however, is the slower rate of recovery, suggesting a more severe atelectasis or perhaps a greater degree of edema formation and increased diffusion difficulty.

In comments at the Princeton Conference on Minimum Ecological Systems, Bjurstedt pointed out that in the G_z vector saturation can fall to as low as 40%, and that desaturation becomes more pronounced when the subject is wearing a G-suit. This is presumably due to retention of blood in the pulmonary circulation and perhaps to the action of raising the diaphragm and intrathoracic organs. Below $3.5G_z$, no desaturation is observed.

Wood [194] (1963) exposed a series of subjects to G_x acceleration on a Mercury couch with hips flexed to 100° on torso and knees 100° on thighs. Although in this position the accumulation of blood in the pulmonary circulation should be less than when the legs are extended, no appreciable difference was in fact observed.

Following up the work of Watson and Cherniack [174] (1961) on the effects of positive-pressure breathing on transverse ($+G_x$) acceleration, Reed et al.[139] (1964) measured arterial oxygen saturation by way of a Waters cuvette connected to a needle in the brachial artery of their subjects. The eight subjects, all experienced in centrifuge work, were exposed to $+7G_x$, $+8G_x$, $+9G_x$, and $+10G_x$ acceleration on a nylon net couch with a back angle of 6°. Acceleration was programed so that peak G was reached in 12 seconds. Each of four different atmospheric situations was explored: air breathing at ambient pressure, air breathing at positive pressure, oxygen breathing at ambient pressure, and oxygen breathing at positive pressure. Pressure was metered at

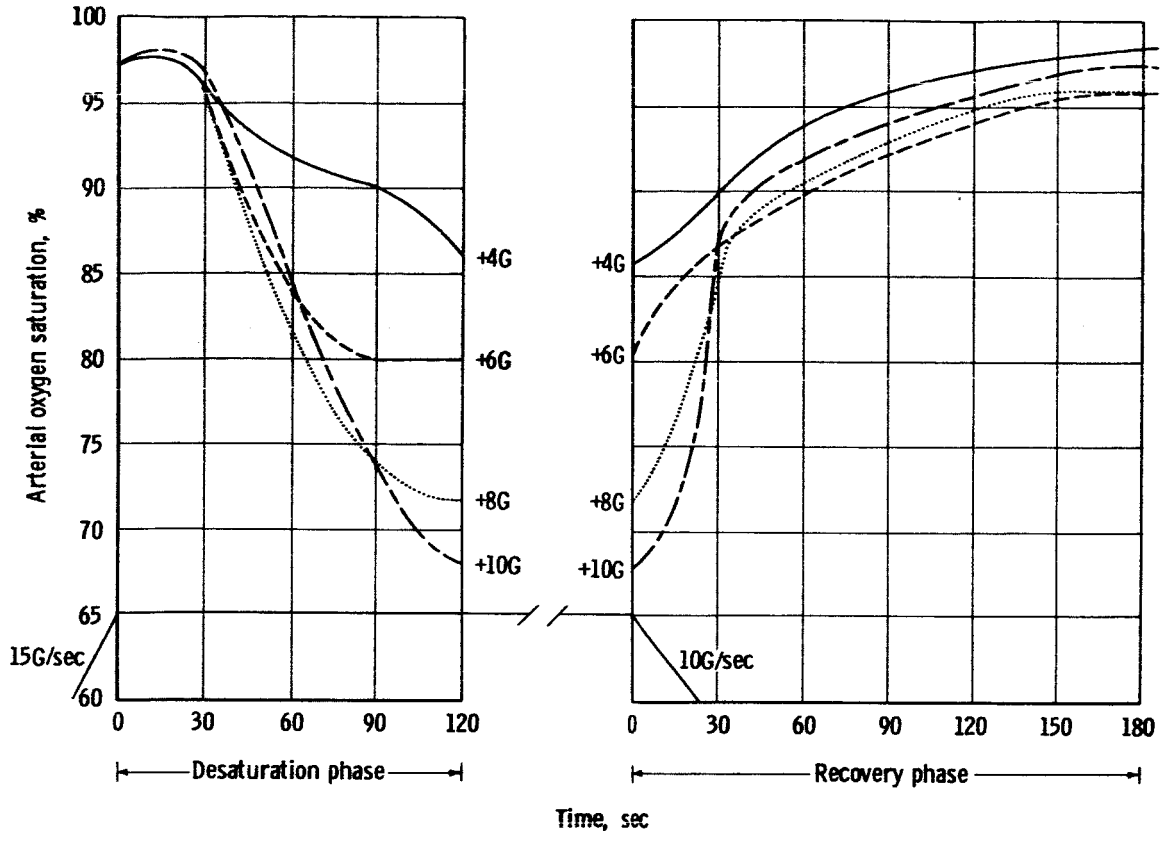


FIGURE 13.—*Arterial saturation of 25 subjects breathing air at 14.7 psia.* $\Delta V = 37{,}000$ *ft/sec.* (Alexander et al.,[3] 1964.)

3 mm per G above ambient by means of a modified full-pressure helmet.

The results of the air breathing runs showed that with or without pressure the saturation dropped about 3% every 10 seconds as compared with a prerun control, until after about 80 seconds the saturation was near the 70% level. Runs were arbitrarily terminated at that point but saturation was continuing to fall at termination.

During the oxygen runs, no fall in saturation was observed in eight runs of less than 100 seconds' duration regardless of the acceleration level. In two runs, one at ambient pressure and the other under positive pressure, where the duration was more than 100 seconds, the saturation fell from 99% to 93%. Since one of these runs was at $+7G_x$ and the other at $+9G_x$, the time factor would appear to be more important than the G-load under these circumstances. Thus, because of the relatively short durations of the runs, these findings do not necessarily indicate that oxygen breathing under positive pressure will counteract the desaturation produced by acceleration. Further work is indicated.

However, the fact that positive-pressure breathing of air does not affect the fall in saturation is interesting. It indicates that in spite of the improved pulmonary ventilation observed by Watson and Cherniack[174] (1962) the ventilation, perfusion, and diffusion relationships must be so altered by the concomitant shunts that adequate gas exchange cannot take place.

Arterial desaturation, of course, results in tissue desaturation. An interesting example is reported by the Russian workers Kovalenko et al.[106] (1963) who measured oxygen

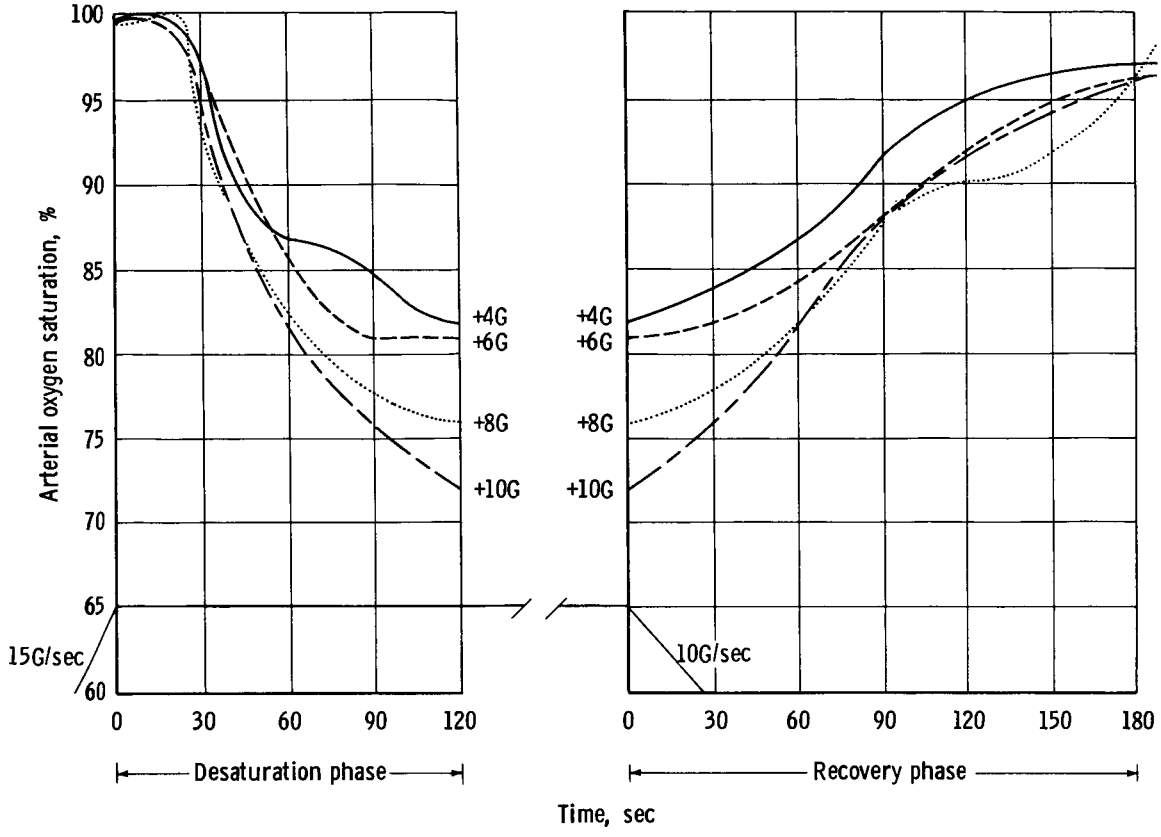


FIGURE 14.—*Arterial saturation of 25 subjects breathing 100% oxygen at 5 psia. $\Delta V = 37{,}000$ ft/sec.* (Alexander et al.,[3] 1964.)

tension directly with platinum electrodes preimplanted in the brain of dogs. They used a polarographic method to determine the relative change in oxygen tension during $+G_x$ acceleration as compared with tension at the start of acceleration. They found that the oxygen tension in the brain of unanesthetized dogs varied with the duration and magnitude of acceleration, with the position of the head relative to the trunk, and individually with different dogs.

At $+2G_x$ to $+4G_x$, Po_2 varied little from the control. With establishment of a $+6G_x$ plateau (duration unstated) it decreased to 90% of control. At 8, 10, and $12G_x$ it became 86%, 81%, and 76% of normal, respectively. During the onset of acceleration to 2, 4, and $6G_x$ an actual increase above starting level was noted. In anesthetized animals a similar increase was not observed. The authors imply that the increase is associated with compensatory measures abolished by the anesthetic.

Cardiac Response

Reference has previously been made to the cardiac response to forward acceleration, although it has not been discussed. Investigations have been made by several groups, including Lindberg [115] (1962) and his colleagues with the Mayo centrifuge, Wood et al.[195] (1961), also with the Mayo centrifuge, Steiner et al.[166] (1960) with the Wright Air Development Division centrifuge, and Hershgold and Steiner [95] (1960) with the same centrifuge.

All are agreed that the cardiac output is either unchanged or, if anything, slightly increased, at least to +5G in man (Lindberg et al.[115] 1962). Prolongation of exposure to

10 minutes does not produce any change beyond that observed initially. The stroke volume appears to remain constant, at least to $+5G_x$ and for short durations. There is a suggestion, however, that it may fall off with prolongation to 10 minutes' duration (Lindberg et al.[115] 1962).

Thus, maintenance of cardiac output depends largely on the pulse rate. This has been observed to vary. In Hershgold and Steiner's study[95] (1960), heart rate in dogs was increased under exposures of 4 and $6G_x$ for 15 seconds; Wood et al.[195] (1961) observed no change in man at 2, 3, and $4G_x$, while Steiner et al.[166] (1960) observed a decrease in dogs at 6, 10, and $14G_x$ for 10-minute periods. However, in a further study, Steiner and Mueller[164] (1961) measured cardiac rate in six human subjects at $8G_x$ for 20 seconds and observed that with the head elevated the heart rate increased 20%; with the head lowered the heart rate decreased 16%; and with the head in the neutral position there was no change in the heart rate. Thus, in the $+G_x$ position the relative change in heart rate appears to depend upon the position of the carotid baroreceptors in relation to the position of the trunk, presumably due to alteration produced by acceleration on the perfusion pressures in the carotid arteries.

Lindberg et al.[115] (1962) observed systematic increases in mean aortic pressure and in right atrial pressure, each of which was also noted by Wood et al.[195] (1961). Surprisingly, Hershgold and Steiner[95] (1960) found a slight decrease in arterial blood pressure in their dogs. This may be related, like heart rate, to the relative head position, or perhaps, as the authors point out, to the technique of measurement, whereby blood pressure was recorded in a femoral artery placed with reference to the supposed position of the heart. Movement of the heart under acceleration could, because of the differences in hydrostatic pressure, produce significant differences between the mean aortic pressure and the arterial pressure as measured.

With respect to other manifestations of hydrostatic effects, petechial hemorrhage in unsupported dependent regions has been reported at 6G in man (Bondurant et al.[19] 1958). Lindberg and Wood[117] (1963) state that visual or cerebral symptoms are not usually associated with transverse accelerations of up to 20G. Again, however, this figure depends upon the position of the head relative to the vector of acceleration. Thus, with subjects restrained in a contour couch such as is used in current space flight, with a head angle 12° up from the horizontal, Chambers[34] (1961) notes reduced peripheral vision at accelerations as low as $9G_x$ in some subjects, loss of peripheral vision and dimness of central vision at 12G, and recurrent complete loss of vision at 15G. Lindberg and Wood's figure of 20G appears much too high.

Marukhanyan[123] (1961) in Russia made ECG recordings with standard leads, a unipolar lead from the left leg and unipolar chest leads, on dogs exposed to $+8G_x$. He found an enlarged P-wave in lead III and the unipolar limb lead, deviation of the electrical axis to the right, low-voltage R and T in the chest leads, and an enlargement of S and Q. Marukhanyan interprets this to indicate an increased pulmonary artery pressure with right ventricular and auricular preponderance resulting from compression of thoracic contents and a shift in the anatomical position of the heart.

However, the main problem in forward acceleration is respiratory. A great deal remains to be done in (a) determining the regional changes that occur in ventilation, perfusion, and diffusion; (b) elucidating further the changes that occur during reestablishment of the equilibrium between 1G and higher acceleration plateaus, and in particular the alteration in gas exchange that takes place during this time; (c) determining the additional work of breathing under different conditions of acceleration; (d) developing more definitive studies on the extent of arterial oxygen saturation; and (e) observing in all these factors the influence of posture, rate of onset, duration, and restraint systems. In fact, the whole field is still wide open.

BACKWARD ACCELERATION ($-G_x$)

Little work has been done on backward acceleration, probably because forward acceleration has been found more profitable in terms of tolerance. Essentially, the problems are the same although respiratory problems are less severe in the $-G_x$ mode.

Rogers and Smedal[140] (1961) undertook a comparison of respiratory changes under forward and backward acceleration to levels of 4, 6, and 8G for 2 minutes with unstated rates of onset. Using a closed-circuit breathing system with a WEDGE spirometer, they measured tidal air, vital capacity, and minute volume with the results shown in table 15. Mean vital capacity and minute ventilation for four of the subjects in the two sets of runs are shown in figures 15 and 16.

These figures are self-explanatory and show a maintenance of vital capacity up to $-6G_x$, and then a fall which is much less than the corresponding fall found in $+G_x$ acceleration. Similarly, there is a marked rise in minute ventilation with $-G_x$ accelera-

TABLE 15.—*Summary of data on five subjects exposed to acceleration stresses.* [Rogers and Smedal,[140] 1961.]

Subject			Volume per unit body surface area, ml/m²	Eyeballs in				Eyeballs out			
Height, cm	Weight, kg	Body surface area, m²		1G	4G	6G	8G	1G	4G	6G	8G
174	64.0	1.75	Tidal	382 286 317	286 308	208 298 277	154	357 361	325	381 290 330	693 320
			Vital capacity	1,657 1,886	543	486	— —	2,114 1,800	1,571	1,371	—
			$\dot{V}$	9,100 5,400 6,300	6,900 5,900	4,300 6,300 5,800	3,700	7,500 7,000	6,200	8,000 7,800 8,000	18,000 7,700
182.9	86.2	2.08	Tidal	421 267 409	433 209	159	216	337 438 582	418 337	499 244	364 346
			Vital capacity	1,635	721	298	—	1,779	1,654	952	—
			$\dot{V}$	5,100 5,700	4,900	3,200	5,400	4,000 6,100 8,700	5,000 5,100	8,000 4,400	8,700 7,300
171.5	69.0	1.82	Tidal	453 380	271 494	192 216	—	308 293	408	405 488	576
			Vital capacity	2,005 1,566	714	302	— —	1,538 1,264	1,566	1,264	—
			$\dot{V}$	5,700 5,000	4,300 4,900	3,600	— —	4,800 5,500	6,100	7,300 8,100	10,900
189.9	69.0	1.95	Tidal	449 413	462 364 390	286 285	288	486 357	1,123	782	—
			Vital capacity	1,897	1,462	1,103	—	1,949	2,000	1,667	—
			$\dot{V}$	5,000 6,300	3,900 4,400 5,500	3,700 4,000	4,600	4,900 4,300	12,400	10,200	—
175.3	84.0	2.0	Tidal	460	275	—	—	422	532	—	—
			Vital capacity	1,150	550	—	—	1,350	1,400	—	—
			$\dot{V}$	9,700	1,050	—	—	10,100	14,400	—	—

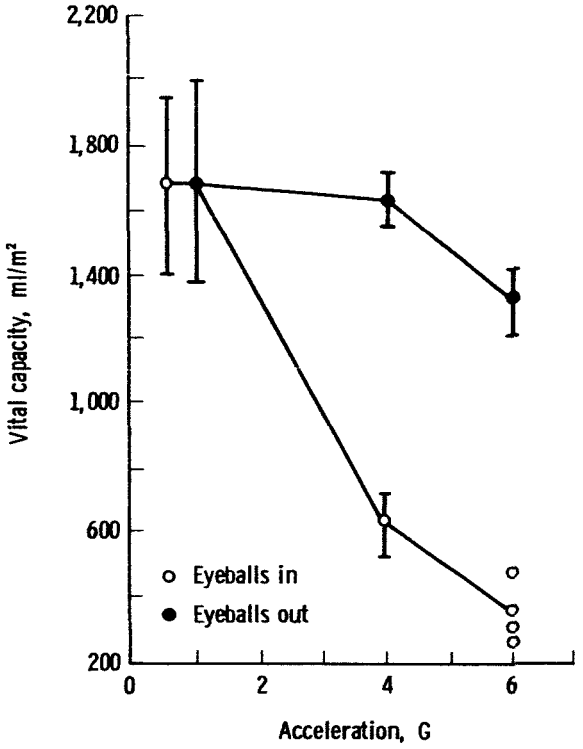


FIGURE 15.—*Vital capacity, mean ± standard deviation for four subjects.* (Rogers and Smedal,[140] 1961.)

tion. This respiratory "drive" under backward acceleration has been commented on previously and its actual cause is debatable. However, in the $-G_x$ situation, because of freedom of movement of the anterior rib cage and abdomen, it can be indulged and does not cause the stress observed in $+G_x$ acceleration.

Effects of backward and forward acceleration on respiration were studied by Smedal et al.[155] (1963). While functional residual capacity in two subjects under $+G_x$ acceleration was reduced from 2.9 and 2.6 liters, respectively, to 0.6 liter at $+4G_x$ and 0.9 liter at $+6G_x$, there was no change at $-4G_x$ and an actual increase of 0.4 liter at $-6G_x$.

The question of oxygen uptake was examined in the same study. However, since only the single statement is made that oxygen uptake increased to 264 ml/m²/min at 4G and 300 ml/m²/min at 6G, there is little basis for comment.

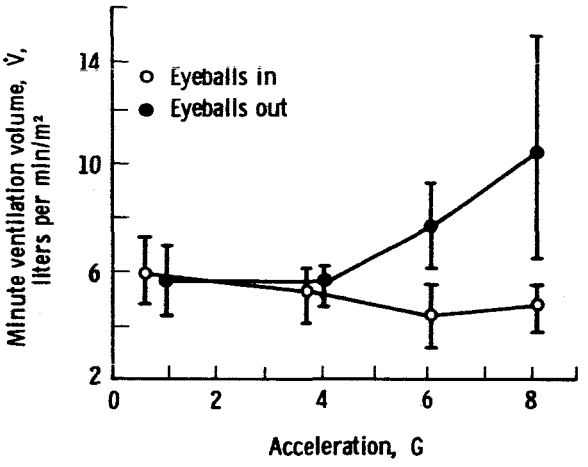


FIGURE 16.—*Minute ventilation volume, mean ± standard deviation for four subjects.* (Rogers and Smedal,[140] 1961.)

A major problem in backward acceleration noted by Smedal et al.[154] (1960) and Creer et al.[54] (1962) is blurring and disturbance of vision. Although suggestions have been made that this might be caused by displacement of the lens or tilting of the retinal receptors, Smedal et al.[155] (1963) in a comprehensive study showed that while visual acuity was impaired, there was no astigmatism or corneal distortion. Disturbance of vision was therefore probably occasioned by excessive lacrimation and the presence of tears on the cornea.

In this same study, other physiological parameters were explored. Under backward acceleration blood pressure showed a characteristic increase, as measured on the arm at heart level, from 120/80 to 150/115 at 8G. Since no account was taken of the relative displacement of heart and arm under acceleration, these figures are not too meaningful. A wide variation in heart rate was found, from bradycardia to tachycardia, perhaps dependent on the relative position of the head.

Arterial saturations in backward and forward acceleration as measured by ear oximeter are compared in figure 17. Allowing for the deficiencies of ear oximetry, there is relatively much less desaturation in the $-G_x$ vector. This is an interesting finding in

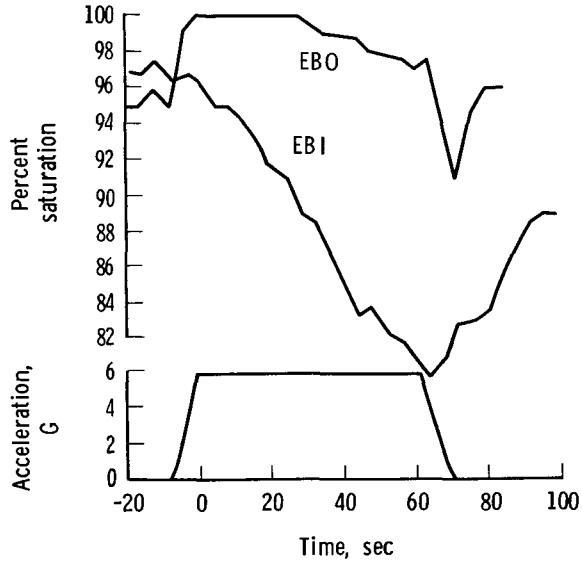


FIGURE 17.—*Human arterial hemoglobin saturation during acceleration.* (Smedal et al.,[155] 1963.)

itself, about which one can only speculate at this time. Perhaps it is related to the fact that in $-G_x$ acceleration the heart and major vessels do not contribute to any pressure on the lungs. No other cardiovascular studies appear to have been carried out during acceleration in the $-G_x$ vector. Smedal et al.[155] (1963) also observed petechiae in the forearms, inability to fuse a double image, and graying of vision at $-8G_x$.

Although subjects are unhappy with the $-G_x$ vector, despite easier breathing, and although it is unlikely to be used as a position of preference, it is obvious that for completeness of data much of the investigation previously undertaken in the $+G_x$ vector should be repeated for the $-G_x$.

LATERAL ACCELERATION ($\pm G_y$)

If little has been done in the investigation of backward acceleration, even less is reported on lateral acceleration. Such effects as have been described are related to increase in hydrostatic pressure and vascular engorgement.

Clark[42] (1961) exposed subjects to 3, 4, and $5G_y$ for 10-second plateaus. Apart from body displacement effects resulting from inadequate restraint, the chief effects observed were petechiae in the right orbit and right temple at $-5G_y$. In one subject a scleral hemorrhage occurred at $4G_y$ with flushing, sweating, and subjective warmth on the same side of the face, and a headache which persisted postacceleration.

The subjects did not report any respiratory difficulty and the author rather casually states that one subject made vital capacity measurements before and after his runs but found no significant change.

Blurred vision in the "outboard" eye was reported in one subject immediately following a $4G_y$ run and persisted for 15 minutes. No pathology or other ophthalmic findings were noted. The cause is obscure.

Headley[91] (1961) confirmed the occurrence of vascular engorgement and pain in the dependent forearm and elbow, along with occasional chest pain and tugging. In addition, he reported that X-ray at $-6G_y$ showed marked displacement of the heart, radio-opacity of the right lung, displacement and flattening of the left hemidiaphragm, although the right appeared to be normal, and blanching of the left lung. From the above, it would apear that ventilation and pulmonary circulation are likely to be considerably disturbed under those conditions.

It is difficult to imagine an operational situation in which an individual might be exposed to prolonged lateral acceleration, unless some future vehicle design dictates a sideways seating arrangement for astronauts. However, if only to establish more complete knowledge, it is considered that the lateral acceleration vector should receive a full investigation.

TUMBLING AND COMBINED ACCELERATIONS

Tumbling became a problem with the development of aircraft ejection seats, and was initially investigated by the Germans during World War II. Weishofer, quoted by Edelberg et al.[64] (1954), spun human subjects without apparent ill effects at 90 rpm with the center of rotation at the center of gravity. Further work was done on monkeys to 120 rpm by Jasper[101] (1947) but more definitive studies became necessary when actual problems began to arise in high-speed ejection.

In the ejection situation, of course, the problem is more than one of simple tumbling, since the tumbling takes place in a decelerative field which may for a short time be as high as 50G. In a space vehicle in a gravity-free state, however, simple tumbling could be a real problem, particularly if the reaction control system should fail after a spin had been imparted.

Edelberg et al.[64] (1954) undertook a thorough investigation of the problem of simple tumbling, using anesthetized dogs lying on their sides with two different centers of rotation—one through the heart and the other 17½ cm caudad of the heart. ECG, respiration, arterial pulse, and blood pressure were recorded along with acceleration and rate of rotation. In some cases end cannulae were used, which tend to give somewhat higher blood pressure readings than actually occur. Rates of rotation ranged to 200 rpm.

In a theoretical analysis the authors point out that movement of blood may be regarded as the result of a pressure gradient along the line of flow. Thus, a centrifugal force directed away from the heart produces an increment of pressure in both the venous and arterial sides of the circulatory system. Flow would continue unabated were it not for the highly distensible venous bed. When pooling in this bed is sufficient, the return of blood to the heart will be inadequate and cardiac output will fall. If this fall produces a pressure drop in the cerebral circulation greater than the increase in hydrostatic pressure, cerebral hypoxia will ensue.

When the center of rotation is moved caudad, the hydrostatic column to the foot is shortened and a lesser degree of pooling can be expected. Conversely, of course, the negative acceleration ($-G_z$) effects on the cerebral circulation can be expected to increase. Movement of the center of rotation cephalad will increase the positive acceleration ($+G_z$) effects. Thus, the final effects are governed both by the rate of rotation and the position of the center of rotation.

In actual experiment, Edelberg et al. found that when dogs are rotated at rates above 140 rpm cardiac output virtually ceases and acute hypoxia ensues. The A–V pressure difference falls to between 5 and 20 mm Hg, respiration is inhibited, tachycardia develops, and oral, ocular, and rectal hemorrhages may occur. The animals, however, generally survive 2 to 3 minutes' exposure to these conditions. Since the hydrostatic pressure increases as the square of the distance from the center of rotation, the blood pressure response is complicated. At 140 rpm, venous and arterial pressures at eye level are of the order of 120 to 140 mm Hg, while at the carotid sinus they range from 50 to 70 mm Hg. Consequently, in the presence of a pressure sufficient to produce oral and ocular petechiae, a tachycardia can arise from stimulation of the carotid sinus. The general effects on heart rate and on arterial and venous blood pressures at different rates of rotation are illustrated in figure 18. At rotation rates above 160 rpm, subatmospheric pressures were observed at the center of rotation.

On cessation of rotation, the sudden return of cardiac output produces arterial pressures at eye level of as much as 400 mm Hg. The authors suggest that the cerebral hemorrhages found in 5 out of 11 autopsied animals are more likely to have been produced by the postrun pressures than by pressures developed during a spin.

Movement of the center of rotation toward the tail produces, as expected, bradycardia, maintenance of respiration as opposed to apnea, higher pulse pressure and A–V pressure difference, and a greatly elevated jugular pressure, as illustrated in figure 19.

The apnea found with the center of rotation at the heart is in contrast to the hyperpnea found in $+G_x$ acceleration and moderate levels of $+G_z$. A possible explanation may lie in the fact that the viscera and the diaphragm move downward with the radial acceleration and may stimulate stretch receptors in the lungs, with consequent inhibition of the inspiratory center. A similar apnea may be observed at high $+G_z$ levels.

The pathology observed postmortem in 11 dogs after rotation at 195 rpm for 1 minute included, in various animals, cerebral hyperemia with microscopic or macroscopic

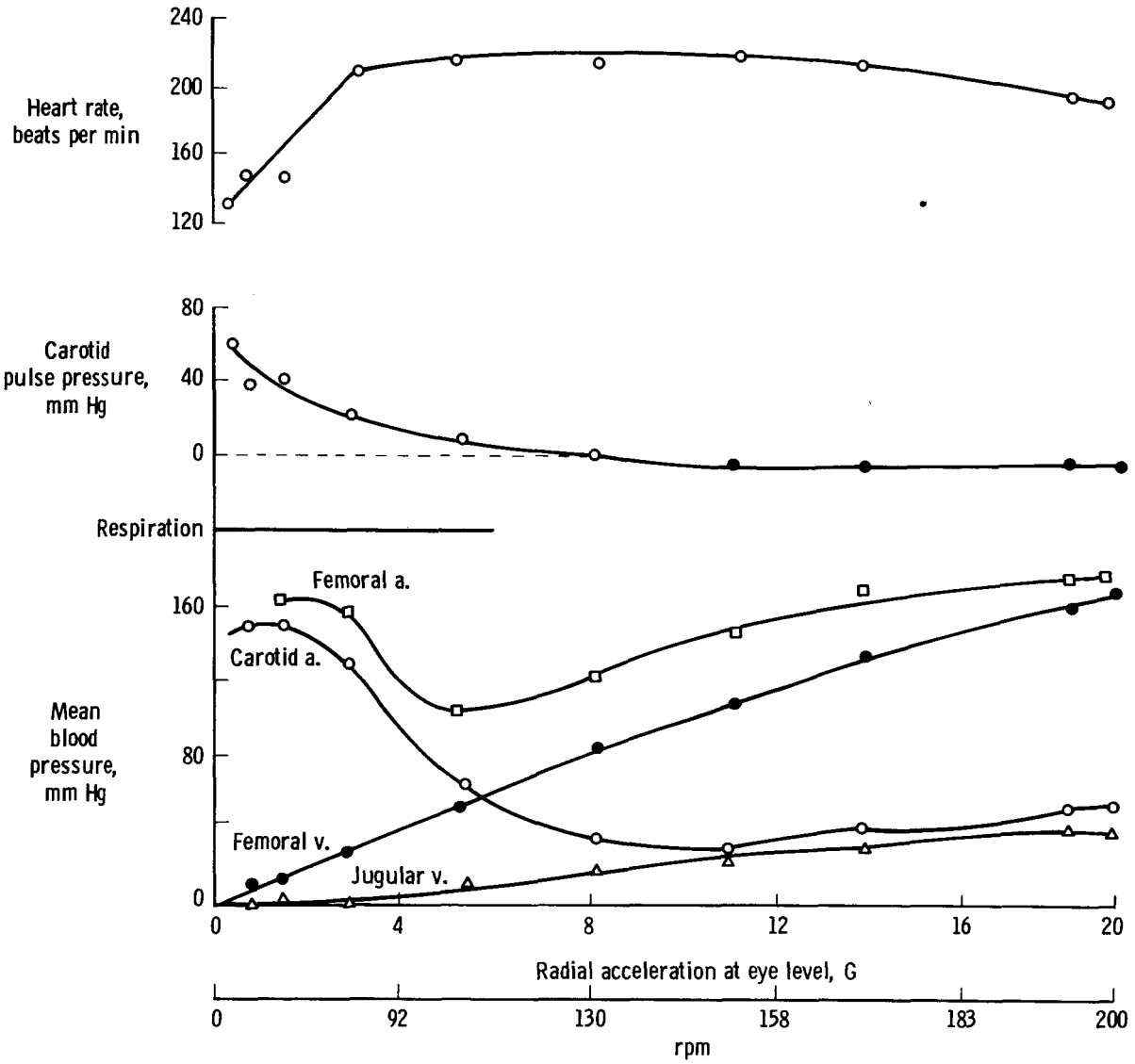


FIGURE 18.—*Effect of spinning a dog about a center of rotation through the heart at various speeds for 30 seconds.* (Weiss et al.,[178] 1954.)

hemorrhage; subendocardial hemorrhage; atelectasis with lung edema, hyperemia, or hemorrhage; hyperemia in the viscera; and rectal hemorrhage.

Weiss et al.[179] (1954) used the same apparatus for studies on humans at rates of rotation up to 125 rpm. Data included ECG, respiration, and blood pressure. In this case, arterial pressure at eye level was determined by an indwelling needle in the radial artery held at a point the same distance from the center of rotation as the eyes. Venous pressure was registered in the antecubital vein and the great saphenous vein at the foot.

Centers of rotation were the heart and the iliac crest, the latter being the center of gravity of a fully equipped man in a conventional ejection seat.

Subjectively, vertigo ceases after the table reaches constant speed. With center of rotation at the heart, 60 rpm is tolerable and even pleasant. Negative acceleration ($-G_z$) symptoms are manifest at 80 rpm and are tolerable at 125 rpm for only a few seconds.

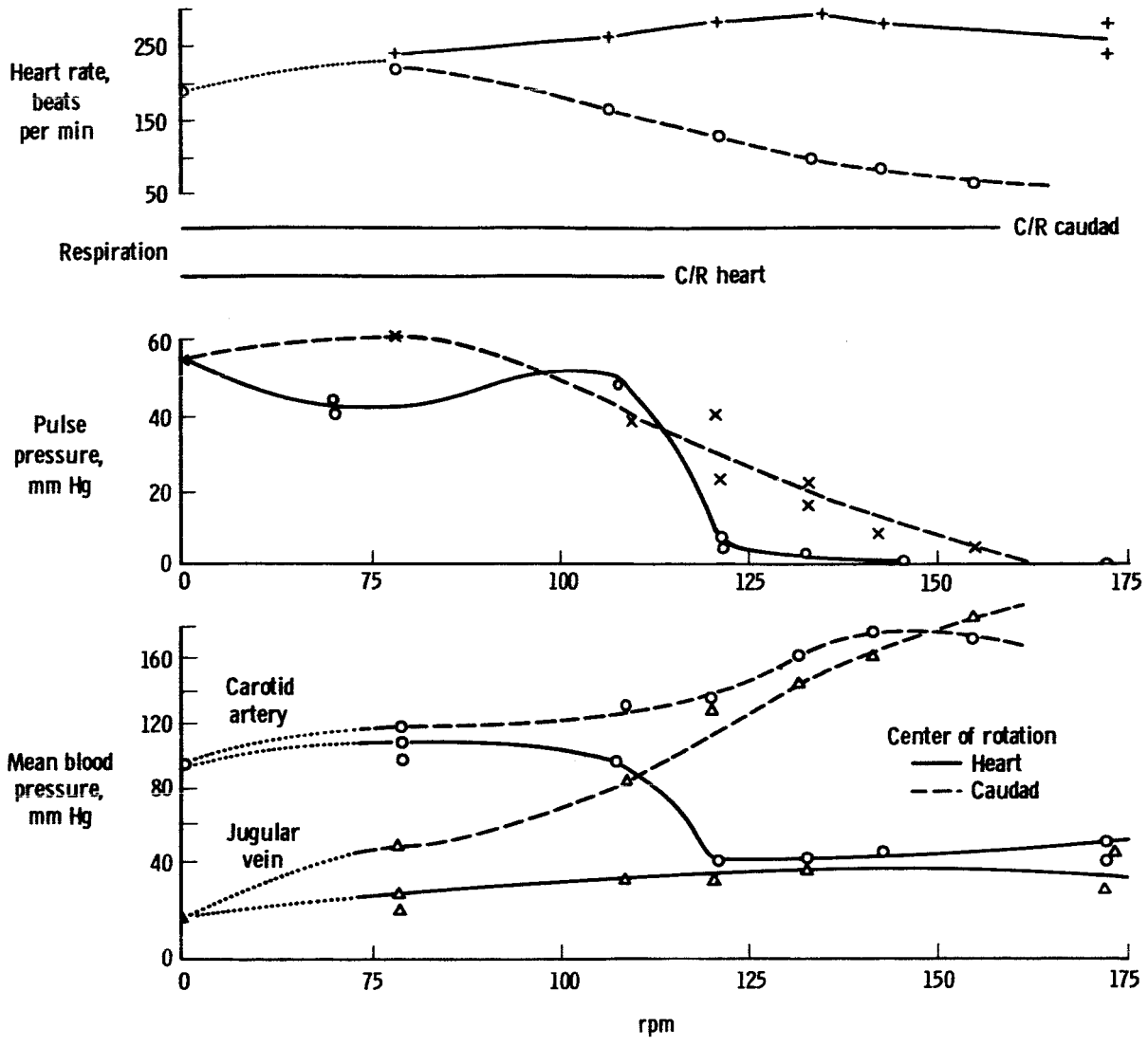


FIGURE 19.—*Effect of change in center of rotation of dog to a point 17.5 cm caudad of heart.* (Weiss et al.,[178] 1954.)

The effects of positive acceleration ($+G_z$), namely, numbness and pressure in the legs, develop slowly, but pain is evident at 90 rpm. No confusion or incipient loss of consciousness is observed, but in some subjects slight spatial disorientation, headache, nausea, or mental depression are noted for several minutes after the run. Repeated runs appear to increase the incidence of postrun nausea, headache, and depression.

With the center of rotation at the level of the iliac crest, the symptoms more closely resemble those of negative acceleration ($-G_z$), with very unpleasant head pressure at 70 rpm. At higher rotation rates, 85 to 90 rpm, the head symptoms approach intolerability, although positive acceleration ($+G_z$) symptoms are unnoticed.

Circulatory effects in humans spun about a center of rotation through the heart are illustrated in figure 20. The relatively greater rise in venous pressure at the foot in human subjects is attributable to the longer hydrostatic column. Respiration is relatively un-

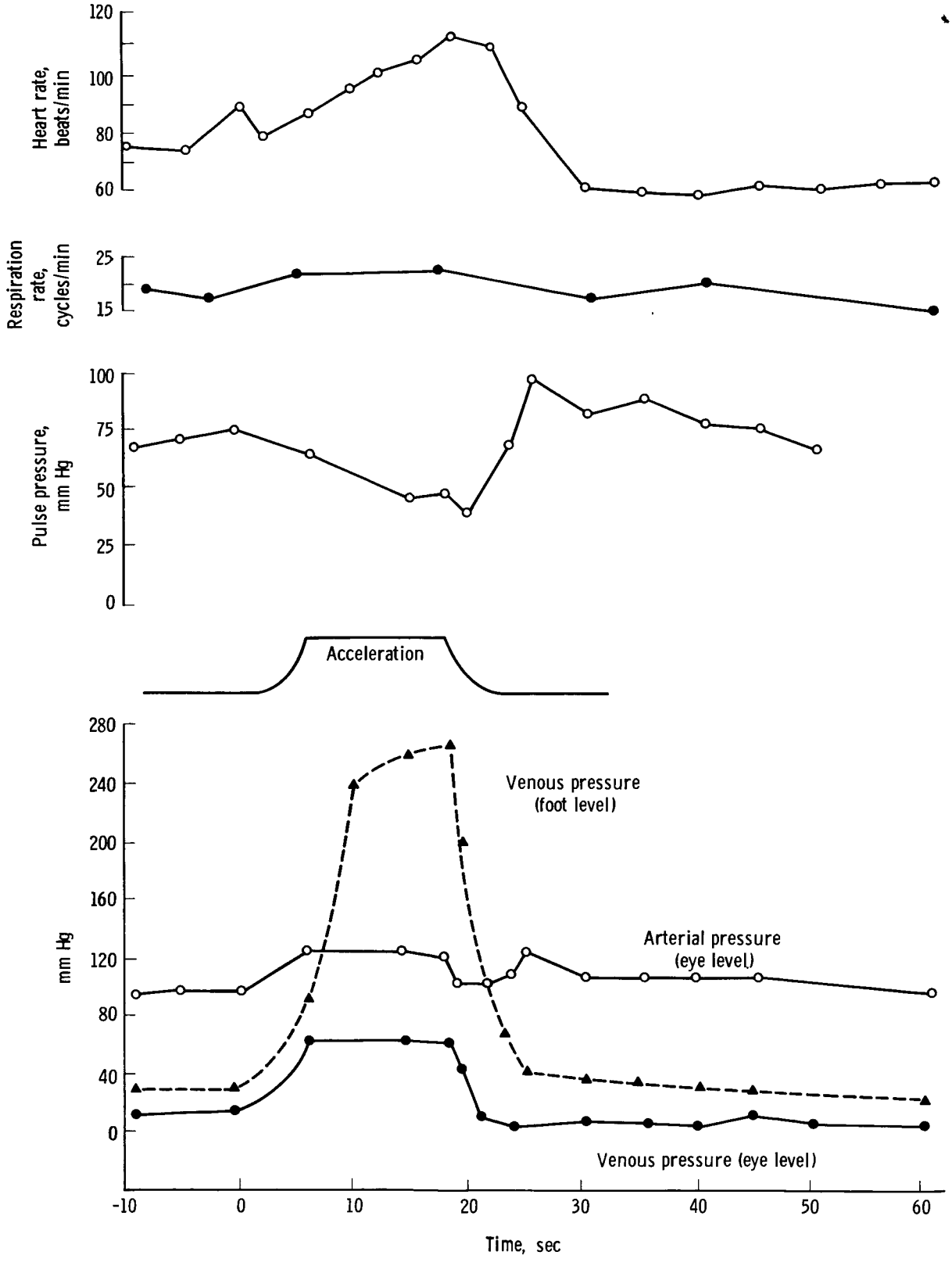


FIGURE 20.—*Effects of spinning a human about a center of rotation through the heart at 106 rpm.* (Weiss et al.,[179] 1954.)

changed; however, it is also unaffected in dogs at the same rate of rotation.

With the center of rotation at the level of the iliac crest there is again a change similar to that in dogs (fig. 21).

Using an extrapolation from the data for dogs, and taking into account the difference in pressures introduced by different lengths of hydrostatic column in man and dog, the authors conclude that unconsciousness from circulatory effects alone would occur in man after 3 to 10 seconds at 160 rpm with the center of rotation at the heart, and at 180 rpm with the center of rotation at the iliac crest.

In this study, conjunctival petechiae occurred during exposures varying from 3 seconds at 90 rpm to 2 minutes at 50 rpm. Petechiae were also found on the dorsum of the foot of subjects who did not wear shoes.

When an acceleration field is added to tumbling, as in ejection, the response is different. It is not merely the summation of deceleration responses and tumbling responses, but, at least at rates of rotation below 100 rpm, the influence of the acceleration

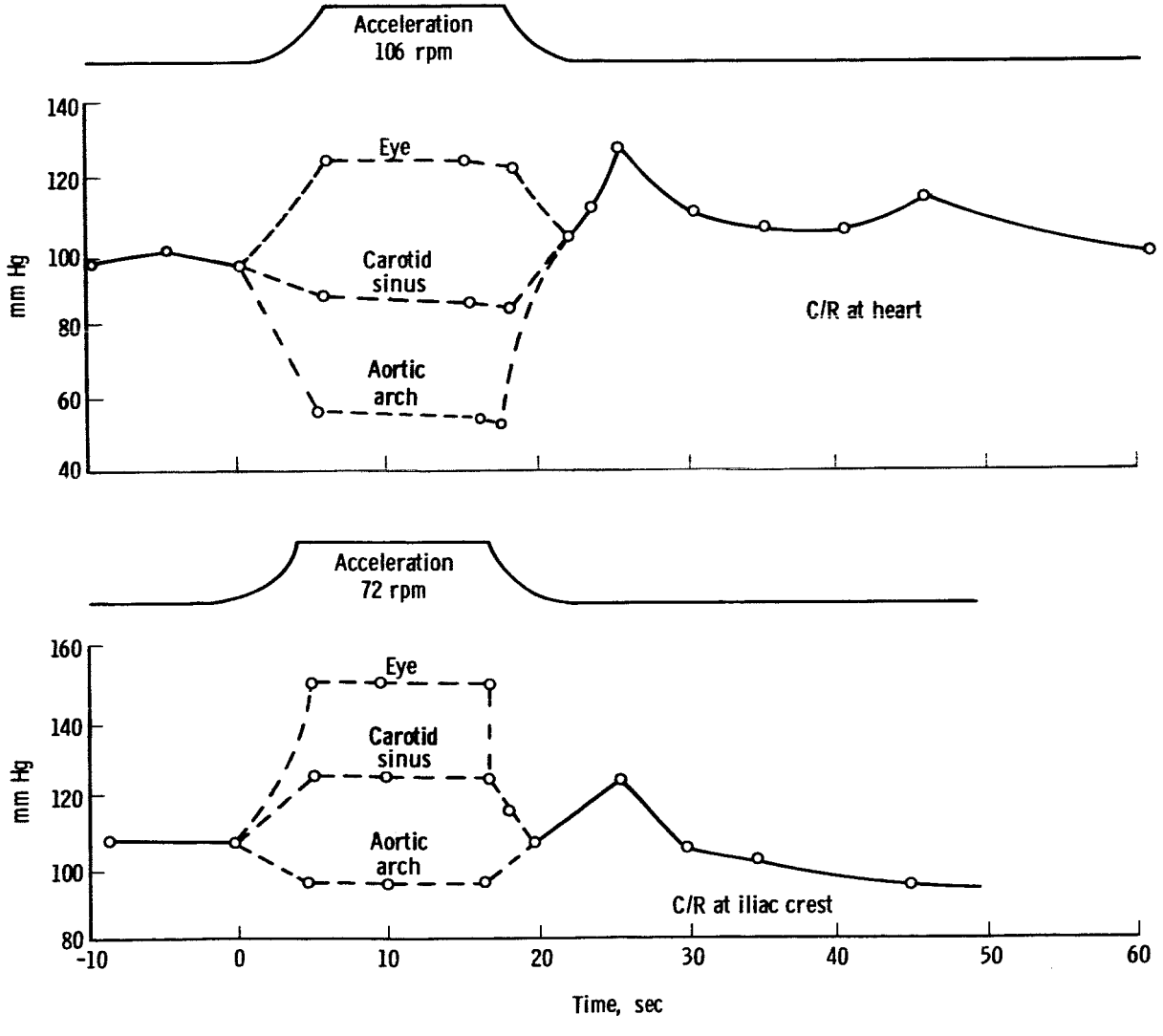


FIGURE 21.—*Mean arterial pressures at various points during rotation of humans.* (Weiss et al.,[179] 1954.)

field is paramount. Thus, when the rotation is in the pitch or yaw plane, the effect resembles severe sinusoidal vibration because of the repetitive oscillatory exposure to positive and negative acceleration. Depending on the impedance and resonance of the body and organs, shear strains will occur and damage may result.

In this regard, Lipkin and Ratcliffe[118] (1954) exposed rhesus monkeys to acceleration forces of 25 or 35G combined with spins ranging from 30 to 150 rpm. Unfortunately, the attitude of the animals in terms of G vector is not clearly stated. Exposures varied from 10 to 30 seconds. Two animals were accelerated, not rotating, to 25 and 35G and then allowed to decelerate while being rotated at 110 rpm. The center of rotation for the animals was about 4 inches below the heart, and the sitting height was about 14 to 16 inches. Postmortem examination, 1 to 6 hours after exposure, revealed tissue damage in the internal organs of all the animals, manifested by vascular congestion, edema, hemorrhage, formation of hyaline thrombi, and separation of parenchymal liver cells.

In an able summary of the physiology of combined acceleration, Edelberg[63] (1961) points out that the cardiovascular response is such that at 200 rpm in simple tumbling, or at $-10G_z$ negative acceleration, engorgement of tissues and vascular rupture may occur in animals, but when the two are combined these effects do not occur, nor do blood pressures attain the theoretical maximum expected from the hydrostatic pressures developed.

DIAGONAL VECTORS

Sustained acceleration applied in a diagonal vector has received almost no attention. Smedal et al.[154] (1960) carried out tracking task experiments at 5.7 to 7G for 1¼ to 6½ minutes in the $+G_z$ and $+G_x$ vectors and in a diagonal between them, during which they measured certain physiological changes previously discussed. Unfortunately they failed to report any findings in the diagonal vector. There appear to be no other studies in which diagonal vectors have been used.

OTHER PHYSIOLOGICAL RESPONSES

Cellular Response

Polis[134] (1961) very rightly observes that acceleration investigations have been dominated by hemodynamic concepts, but that at the cellular level physiological stress may be defined in terms of substrate supply and energy demand. On the hypothesis that under conditions of limited supply the energy distribution could be controlled by hormonal mechanisms, he determined the tolerance of rats to +20G in an unstated axis, in three groups, using as a measure the time taken to reduce the heart rate from 8 to 2 beats per second. One group, having undergone hypophysectomy, showed a 300% increase in survival time compared with control; a second group, after adrenalectomy, showed a 60% drop in survival time; and a third group, after both operations, approximated the normal period. The hypophysectomized group, however, with subsequent atrophy of the adrenal cortex, showed a gradual loss in ability to withstand acceleration stress, although at 15 weeks postoperative their resistance was still significantly greater than that of the controls. These findings are illustrated in figures 22 and 23.

Polis postulates that, with an intact hypophysis, the energy available is so distributed that some is expended on long-term needs such as protein synthesis, fat conservation, and glycogen storage, while some is made available for immediate use. He suggests that hypophysectomy depresses the long-term demands and diverts energy for vital cellular function. Thus, according to his hypothesis, acute acceleration stress is more readily met in the hypophysectomized animal.

To ascertain the nature of any associated substrate changes, he and his colleagues (Polis et al.[136] 1962) determined some metabolic changes in rat brains which were rapidly excised from normal and centrifuged animals and then frozen, weighed, deproteinized with picric acid, and analyzed for free amino acids by ion-exchange chromatography. Decreases greater than 50% were found in beta-hydroxyaspartic acid, serine, urea, and glutathione, along with a large in-

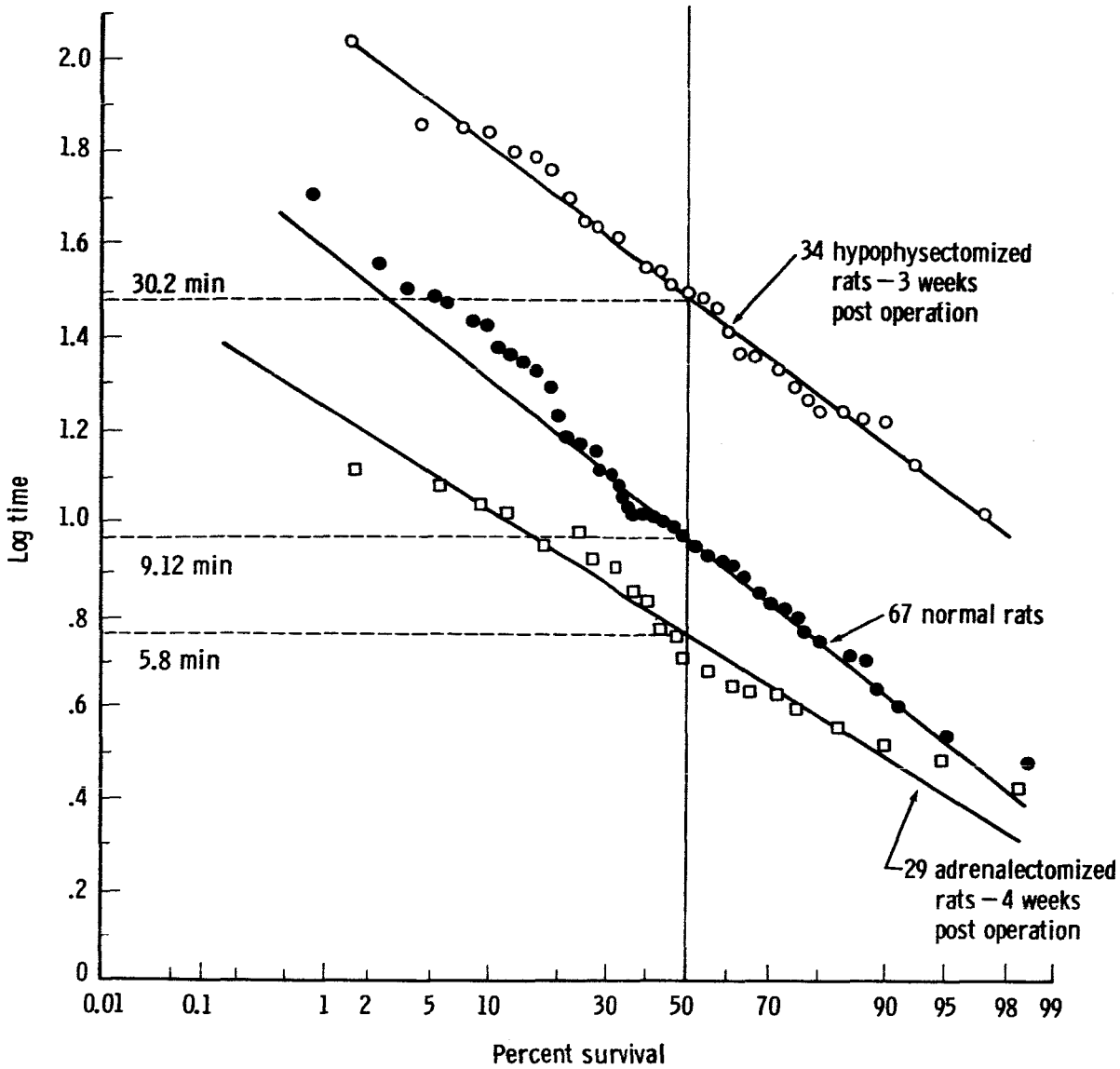


FIGURE 22.—*Probit plot of percent survival vs log time for normal, hypophysectomized, and adrenalectomized rat populations at 20G. All three curves differ significantly from each other by "Student's" test at less than the 1% level for chance occurrence.* (Polis,[134] 1961.)

crease in free ammonia. This may, as the authors suggest, represent a block in the energy-yielding mechanisms from the respiratory enzymes in the mitochondria, but alternative hypotheses can be suggested to explain the findings.

Stiehm[167] (1962) noted that, histologically, conditioning results in hypertrophy of the reticuloendothelial system (RES) and that stimulation of this system involves alterations in endogenous steroid metabolism. Bearing in mind the work of Polis, he postulated a common mechanism operating by way of the hypophyseal-adrenal axis. The resulting specific humoral hormone pattern might permit a readier tolerance of cellular hypoxia. To show that stimulation of the RES would enhance tolerance, Stiehm gave 10 daily consecutive injections of endotoxin to rats in dosage insufficient to produce ill-

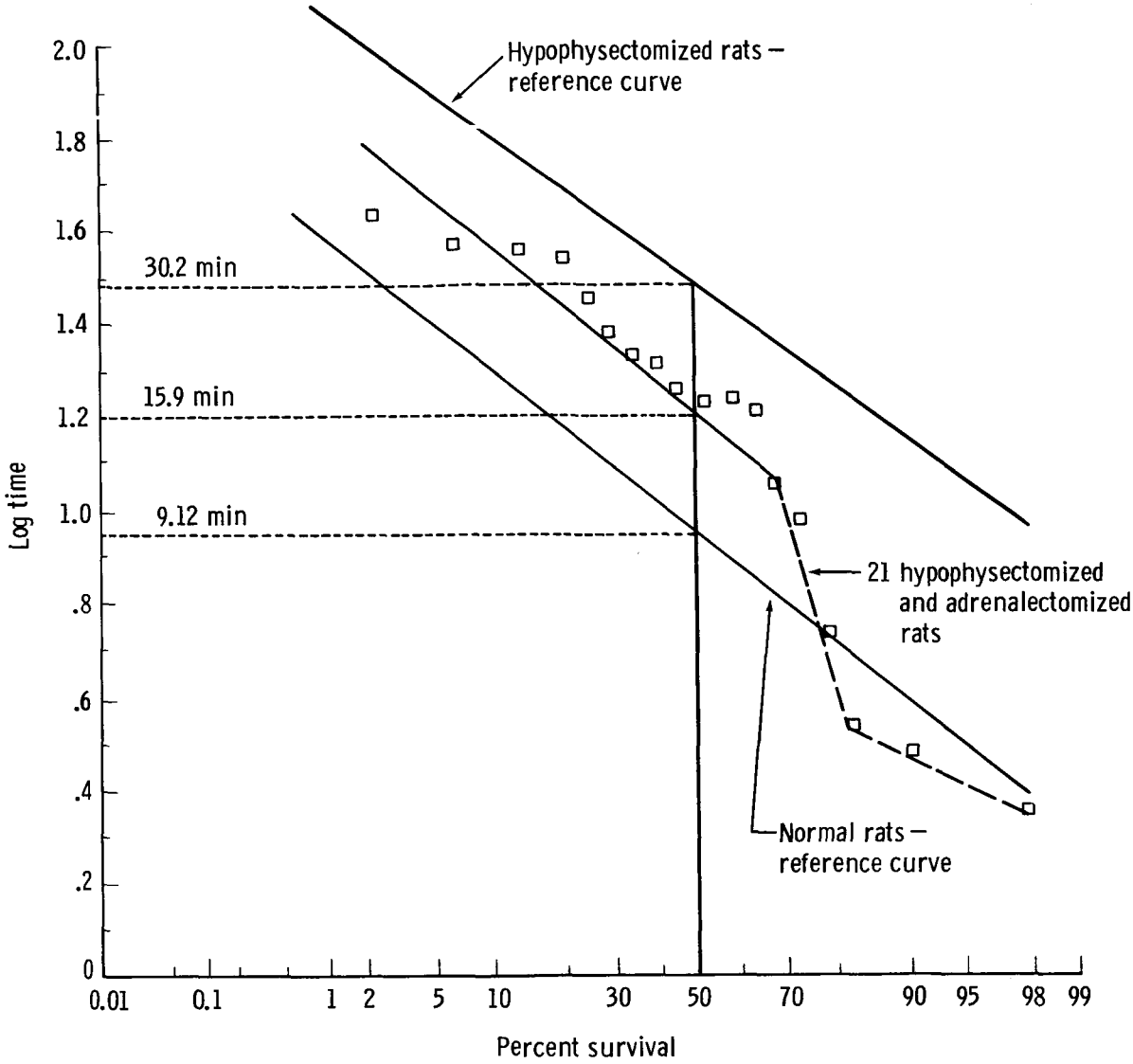


FIGURE 23.—*Effect of hypophysectomy and adrenalectomy on the resistance of rats to acceleration at 20G.* (Polis,[134] 1961.)

ness and found that the median survival time for exposure to $20G_z$ was increased from 9.7 to 14.2 minutes. The survival time of another group rose from 11.3 to 23.6 minutes.

It would thus appear that there is a cellular response to acceleration stress which has been little studied. This work should be pursued.

Effect on Renal Output

The question of maintenance of blood volume raises some interesting considerations. In a comprehensive review of the subject, Gauer et al.[80] (1961) discuss the evidence for cardiorenal control of blood volume mediated by the antidiuretic hormone of the pituitary. Henry et al.[93] (1956) indicate the presence of volume receptors in the right and perhaps the left atrium, stimulation of which inhibits the secretion of antidiuretic hormone (ADH) and causes diuresis.

H. L. White et al.[182] (1926) showed that assumption of the erect posture after recumbency will reduce the rate of urine secre-

tion. Assuming that the effect is due to a reduced atrial volume, a similar reduction would be expected under applied $+G_z$ acceleration. Although Noble and Taylor[129] (1953) failed to demonstrate the presence of ADH in the urine of subjects exposed to blackout levels, Stauffer and Errobo-Knudsen[162] (1953) showed that when accelerations of $+3G_x$ and $+5G_x$ were applied for 1 minute, there was a significant reduction in urinary output during and after acceleration in fully hydrated subjects. This paper was criticized by Garrow[76] (1960) on theoretical grounds, but this fact does not detract from the findings illustrated in figure 24. In Clark's study[40] (1960) of a subject accelerated at 2G for 24 hours, it is interesting to note that although the fluid input was 2,250 cc the urine output was only 890 cc.

On the other hand Watson and Rapp[176] (1962), using a rigid protocol, exposed fully hydrated subjects to $+4G_x$ after establishing preacceleration controls, and measured during five subsequent 15-minute periods the mean urine volume per minute (V), osmolal clearance (C_{osm}), free water clearance (C_{H_2O}), glomerular filtration rate (GFR), effective renal plasma flow (ERPF), sodium excretion (U_{Na}), potassium excretion (U_K), serum osmolality (P_{osm}), and osmotic ratio of urine to serum (U_{osm}/P_{osm}). The results are shown in table 16 and figure 25. They indicate an increase in glomerular filtration rate and effective renal plasma flow during and after the stress, along with an increase in urine volume and free water clearance maximum in the second postacceleration period.

Neither of these studies included estimations of ADH. Garrow[76] (1960) measured the output of ADH under acceleration conditions in a series of three sets of experiments,

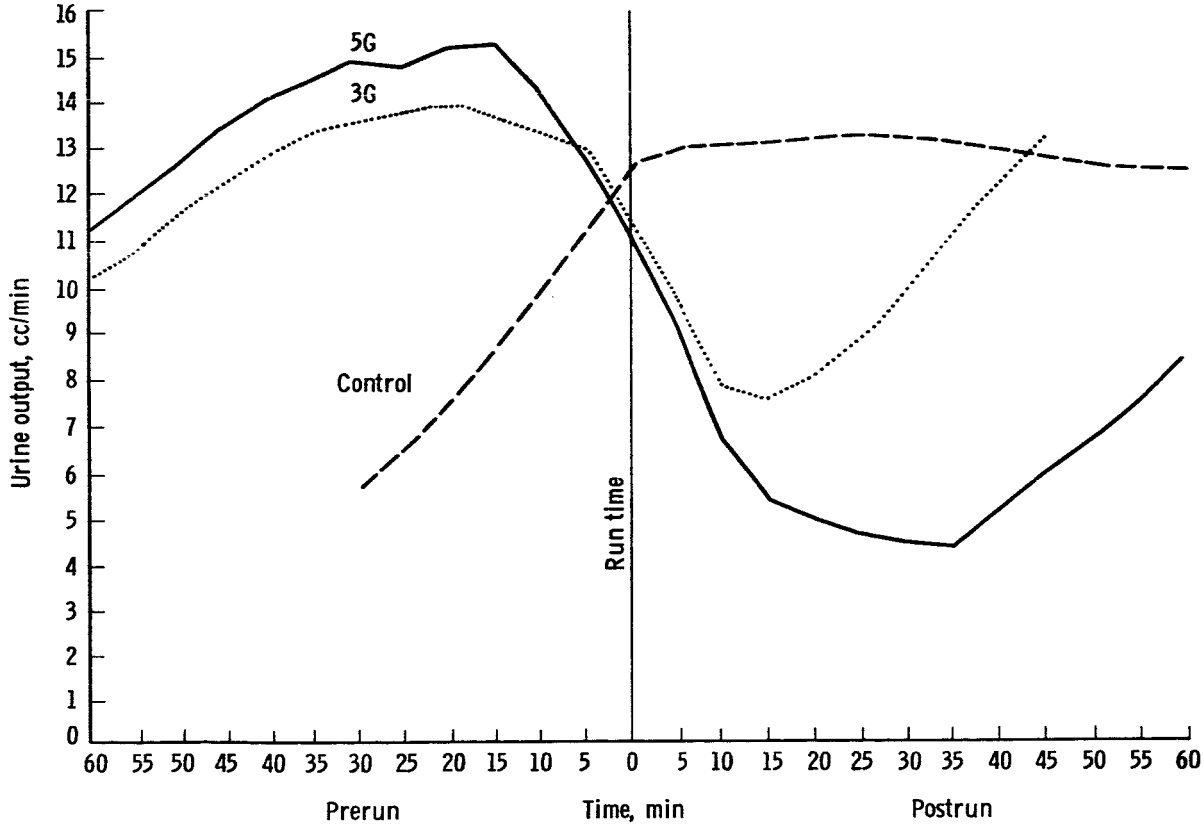


FIGURE 24.—*Urine output before and after acceleration.* (Stauffer and Errobo-Knudsen,[162] 1953.)

TABLE 16.—*Mean renal response to forward acceleration.*[a] [Watson and Rapp,[176] 1962.]

Period[b]	GFR, ml/min	ERPF, ml/min	V, ml/min	C_{osm}, ml/min	C_{H_2O}, ml/min	$\frac{U_{osm}}{P_{osm}}$	U_{Na}, μeq/min	U_K, μeq/min	P_{osm}, mOsm/l
C	112	541	1.86	4.02	−2.16	2.34	324	88	291
4G	[c]124	587	2.01	4.08	−2.07	2.23	350	85	290
1	120	557	2.41	3.79	[c]−1.38	[c]1.90	293	77	292
2	[c]126	[d]637	[d]2.98	3.90	[d]−0.92	[d]1.63	333	78	292
3	120	601	2.27	3.67	[c]−1.40	1.93	328	[d]63	292
4	108	552	1.58	[c]3.35	−1.77	2.23	313	[d]56	291
5	116	598	1.61	3.52	−1.91	2.33	329	[d]61	291
S.E.	±5.3	±26.8	±0.292	±0.224	±0.245	±0.207	±24.2	±6.3	

[a] Values corrected to 1.73 sq m surface area.
[b] C=mean of four basal periods; 4G=period in which subject was centrifuged at 4G for 10 min; others are the immediate five 15-min postrun periods; S.E.=standard error of mean.
[c] $P < 0.05$.
[d] $P < 0.01$.

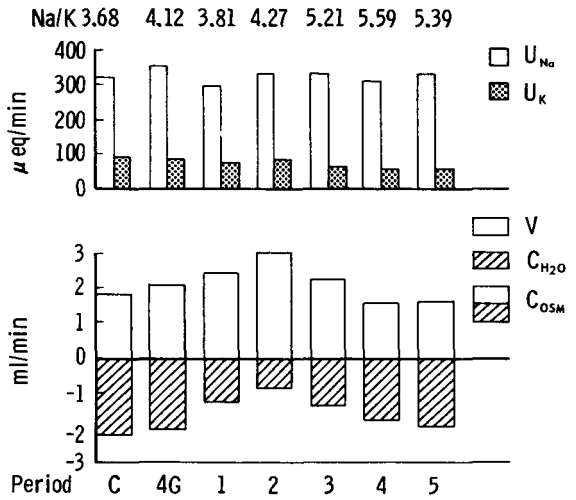


FIGURE 25.—*Renal response to forward acceleration.* (Watson and Rapp,[176] 1962.)

the first two of which were handicapped by the fact that only one subject was used. In the third set, with three hydrated subjects exposed to $+2G_z$ for 10 minutes, the results were equivocal in that one subject always had water retention (11 times out of 11) beginning with the acceleration and progressing for about an hour, one subject did not (0 times out of 2), and one subject had on one occasion out of two. The figures for the latter two subjects are too small to have any significance despite the author's facile arguments, except to indicate that antidiuresis does not always occur at $2G_z$. However, water retention when it occurred was accompanied by an increase of urinary ADH, and although retention did not occur on all occasions of acceleration, adequate function of the ADH mechanism in all subjects was demonstrated by injection of pitressin. To explain the lack of retention, Garrow postulated somewhat unconvincingly, without presenting any evidence, that under acceleration both ADH and adrenalin may be produced, and if the latter is released first it will inhibit the ADH. It is more likely that the $2G_z$ stimulus was inadequate. However, it would seem that with adequate $+G_z$ stimulus there is a water retention, and with $+G_x$ stimulus there is a diuresis.

The explanation of these results must remain somewhat speculative but they are no doubt related to stimulation of a volume-sensitive receptor mechanism. A volume receptor mechanism located in the left and perhaps right atrium and terminal pulmonary veins must of necessity be sensitive to pressure, and it has been shown by Wood et al.[195] (1961) that at $+4G_z$ the right atrial pressure is decreased to −10 to −15 mm Hg, with an even greater decrease in the esophageal (intrathoracic) pressure. Consequently, in $+G_z$ acceleration a pressure-volume receptor would receive a negative stimulus and promote retention.

In the case of the G_x vector, Wood et al.[195] (1961) showed that the right atrial and intrathoracic pressure was considerably increased. This in turn would provide a positive

stimulus and promote inhibition of ADH with consequent diuresis.

Although many tilt-table experiments have been carried out, demonstrating a reduced output of urine on rising from the supine to the passive vertical, it is surprising that little or no investigation seems to have been made of the effect on secretion of tilt to $-1G_z$. This would appear to be a fertile field for further study.

Exercise and Fatigue

Another physiological matter that deserves some examination is the marked fatigue that is commonly observed postacceleration. At best, fatigue is a vague concept physiologically, although certain biochemical changes are associated with exercise. Slonim [152, 153] (1960, 1961) compared the effects of exercise, mock stress, and acceleration in terms of biochemical changes. Five subjects were exercised on a treadmill at 8% grade at a rate of 3.5 mph for 10 minutes. The same subjects were each exposed to a different G-time aggregate, varying from 3G for 30 minutes to 12G for 1.25 minutes, and also to 6G for varying duration, according to their tolerance. Thus the end-points were subjectively established, and it is difficult to equate the amount of exertion imposed during acceleration with that of the exercise.

Measurements were made, among other things, of plasma bicarbonate, blood glucose, phosphorus, creatinine, and urine creatinine, under control, test, and post-test situations. Exercise resulted in a marked decrease in bicarbonate, a rise in blood creatinine, and a drop in creatinine clearance. The only consistent change after acceleration was a slight rise in blood creatinine.

Thus, the allegedly comparable subjective fatigue cannot readily be explained in terms of muscular activity. However, as noted, there is some doubt as to whether the situations are actually comparable, although there is no doubt that postacceleration fatigue is very real. This area should have a systematic study.

CHAPTER 4

Tolerance to Sustained Acceleration

In considering the question of tolerance to acceleration stress, one has to examine two forms: subjective tolerance and performance. Bearing in mind the physiological capacity of the individual, it is obvious that, except in the unusually well motivated, the threshold of voluntary tolerance is usually reached before the stress exceeds the physiological capacity; otherwise injury will occur.

The upper threshold of subjective tolerance, then, is the limit of the capacity to endure the physical and emotional discomfort of a stressful environment, while that of performance is the limit of the capacity to perform adequately the requirements of a given task. In general, performance normally declines before the tolerance threshold is reached.

For tolerance studies, which are of necessity subjective, the question of end point is a difficult one in many cases. Meehan [126] (1961) reviewed the matter without, however, offering anything new. In positive acceleration ($+G_z$) the occurrence of grayout, blackout, or unconsciousness can be used as a standard, and in negative acceleration ($-G_z$) redout, if it occurs, is useful. In transverse acceleration or in more complex forms, dyspnea, discomfort, and pain are the limiting features and are not easily evaluated in standardized terms. However, even the threshold of more objective criteria, such as blackout, varies from individual to individual and from time to time in the same individual. Level of consciousness as measured by the EEG may have considerable possibility, as suggested by Squires et al.[158] (1964), and certainly the EEG provides a clear indication of unconsciousness. Loss of photic drive as observed by Duane et al.[60] (1963) in the EEG at grayout levels might be a useful end-point.

Beckman et al.[14] (1961) have attempted another approach. They showed that between 3.5 and 7G_z (i.e., between grayout and blackout), 60 subjects in 400 tests all demonstrated an objectively noticeable limitation in ocular motility (LOMA). In some subjects the limitation could be overcome by voluntary effort but the resulting movements were ataxic. While this criterion does not eliminate individual and temporal variability, it at least appears to provide an objective end-point.

Another objective end-point was suggested by Marukhanyan [123] (1961), who observed ST and T changes in the electrocardiogram, most pronounced 10 to 20 seconds before unconsciousness, and other cerebral disturbance in subjects exposed to G_z acceleration. Both of these measures, however, apply only to G_z acceleration. It would be valuable if other objective end-points could be developed for different vectors. This area deserves additional study.

FACTORS IN SUBJECTIVE TOLERANCE

There is no doubt that tolerance varies immensely from individual to individual, and from time to time in the same individual. Although the squat, slightly hypertensive subject tends to have a higher tolerance, no correlation was found by Franks et al.[71] (1945) between duration to blackout or unconsciousness and numerous cardiovascular measures. Similarly, Hyde et al.[99] (1962) found no correlation between duration to blackout during positive acceleration, or respiratory performance during forward acceleration, and an extensive number of anthropometric measurements and physical fitness tests, nor was correlation found between $+G_x$ and $+G_z$ acceleration responses and measurements made during other stressful situations.

Gauer[78] (1950) points out the decrease in tolerance effected by excessive use of tobacco and alcohol, generally in association with the shortage of sleep incurred in the same situation. This was also observed by Franks (personal communication), who makes the interesting comment that a "hangover" on some occasions may actually increase tolerance, presumably from the associated generalized mild edema which may occur with a severe hangover and acts to counterbalance the increased hydrostatic pressure. Dehydration, on the other hand, reduces tolerance.

Both Franks and Gauer also noted the debilitating effects of febrile illnesses of respiratory or gastroenteric type, which produce a decrease in tolerance lasting for several weeks, presumably from disturbance of vascular tone.

The disturbance reported by Brent et al.[20] (1960), resulting from the combined effects of acceleration, hyperventilation, and hypoglycemia, has already been noted. Similarly, a marked decrease in tolerance occurs under hypoxic conditions (Gauer,[78] 1950).

MAGNITUDE OF SUBJECTIVE TOLERANCE

Because of the biologic and diurnal variability it is difficult to establish rigid limits under all circumstances of brief and prolonged acceleration. Any stated limits must be interpreted as approximate.

Figure 26 is a compilation, by the author of the present monograph, from numerous sources of various human experiences of brief and prolonged acceleration. For clarity, the symbols indicating acceleration vector are applied to a vector regardless of the body attitude within that vector. Thus the open triangle used for $+G_x$ data includes situations both where a subject is supine and where his legs or head might be raised. Although not all exposures are recorded, the diagram includes all available extreme exposures. It must also be noted that each data point represents a "plateau" of acceleration and not merely an incidental peak. Consequently, accelerations experienced in dynamic simulation of, for example, spacecraft launch and reentry are not included. The lines are an estimation of the maximum voluntary tolerance of healthy well-motivated men using conventional restraint harnesses, couches, and G-suits, but not water immersion, positive-pressure breathing, airbags, and so on. The figure shows that, exclusive of rates of onset, some areas are G-time limited while others are G limited only. These threshold limits must be considered maximum levels and not ordinary working levels.

It will be noted that between 1 and 1¾ seconds, positive G (G_z) tolerance is apparently G limited at the level of 15G. Between 1¾ seconds and about 10 seconds it is G-time limited, tolerance decreasing with increase in time. Thereafter, another G-limited plateau occurs between 10 seconds and 2 minutes at a level of 6G. Between 2 minutes and 3 minutes 20 seconds there is again a G-time limitation, leveling to a new G-limited plateau at 4½G, which continues for an indefinite time. There may be a still further fall to 3G, which has been experienced for an hour without reaching tolerance threshold.

From the work of Stapp[160] (1955) it would appear that there is a G limit of $25G_x$ at the 1- to 1½-second level. Thereafter, although there are no data points to support the hypothesis, it would seem reasonable to have a G-time limit as indicated, leveling at $+17G_x$ and beginning in the 4- to 5-second region. Thereafter the threshold is G limited at $+17G_x$ to the 2-minute mark, at which point a G-time limitation again occurs, gradually lowering the tolerance to about 5 or $6G_x$ at the 10-minute mark. In this region the data points unfortunately are vague.

Higher levels have of course been tolerated for periods of a few seconds, but not in the form of an acceleration plateau. In the early investigation of contour couches, for example, Collins et al.[52] (1958) reported exposure of two subjects to higher than $+20G_x$ for a few seconds in the form of a haversine peak. With a back angle of 17° from the horizontal, one of the subjects blacked out at $+16G_x$; lowering the back angle to 10° from the horizontal allowed him to continue to $+20G_x$.

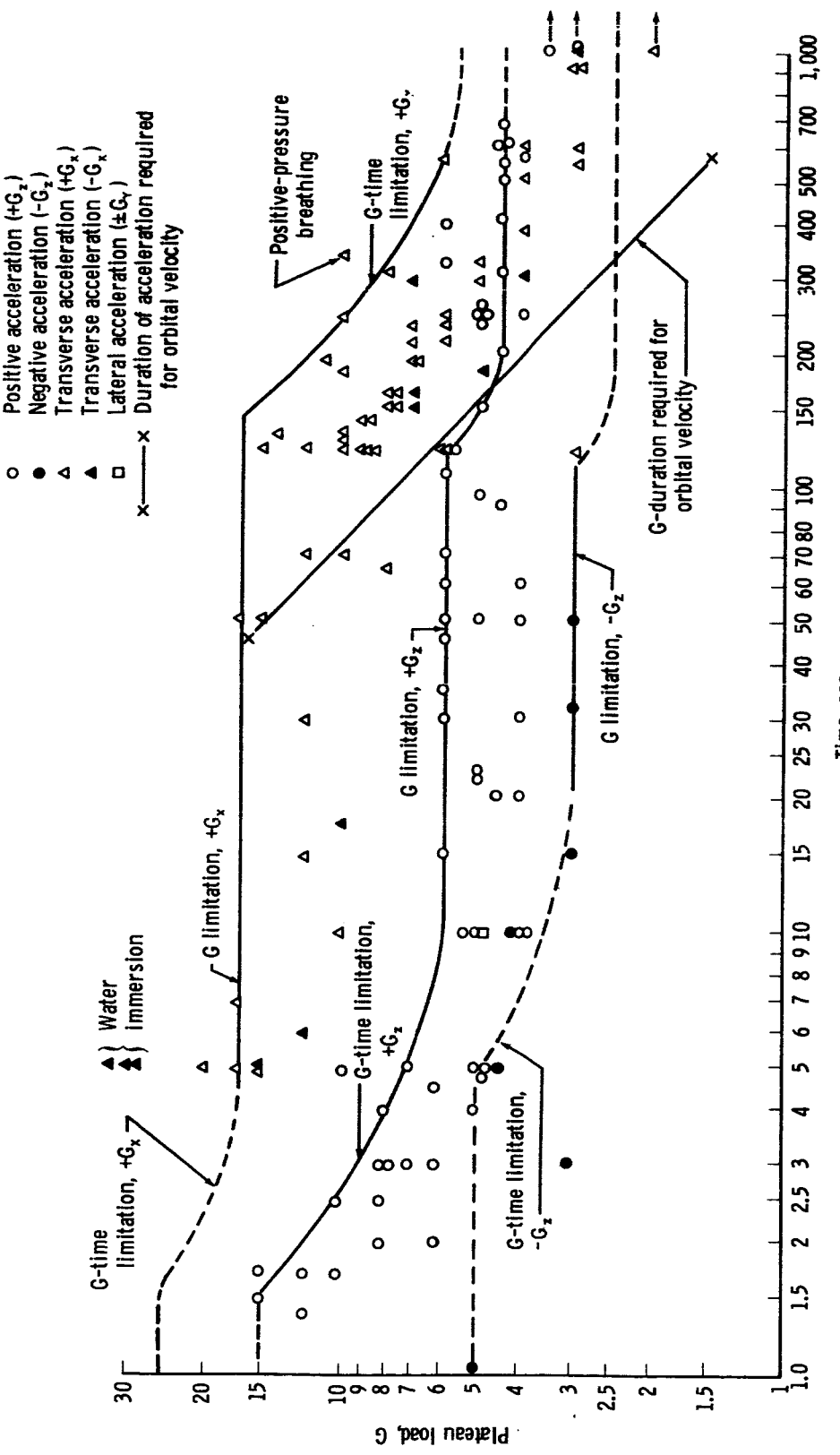


FIGURE 26.—*Human experience of sustained acceleration.* (Data from many sources.)

In a further report (Collins and Gray,[53] 1959) it was noted that one subject maintained $+25G_x$ for several seconds on a molded contour couch with a back angle of 8° from the horizontal. Blackout did not occur, but the subject was unable to maintain air in his lungs.

Data points for negative acceleration ($-G_z$) are very scanty but appear to indicate a G-limited plateau of $-5G_z$ for the first 4 to 5 seconds. This level seems high, since $-3G_z$ for 5 seconds is normally considered to be the tolerance threshold. In view of the data points available, however, it seems acceptable, bearing in mind that this level represents a maximum tolerance. Following this plateau there is a G-time limit reducing the tolerance to $-3G_z$ for at least 50 seconds and probably longer. Whether a further G-time limitation appears is not known.

The shape of the curves on the log-log plot provides an interesting corollary, namely, that we are observing here the failure of different systems with the establishment of new equilibria. Thus, while the interpretation is purely speculative, it may well be that in the $+G_z$ plot we see the effects of hydrostatic pressure on the cerebral circulation between 1¾ and 10 seconds, followed by a different failure at the 2-minute level. Other speculations may be applied to the $+G_x$ and $-G_z$ plots.

It is considered that this type of diagram, where tolerance thresholds are described in terms of plateaus and curves, is a more accurate representation of the true situation than those where the thresholds are represented by straight sloping lines. The results of numerous tolerance studies have been represented in the latter form; for example, see figure 27, prepared by Smedal et al.[154] (1960), and figure 28, prepared by Chambers [35] (1963). Other studies dealing with the establishment of thresholds for plateau G loading are summarized in the annotated bibliography.

A representation of the G-time required to attain orbital velocity is included in figure 26. It will be observed that the stress is readily

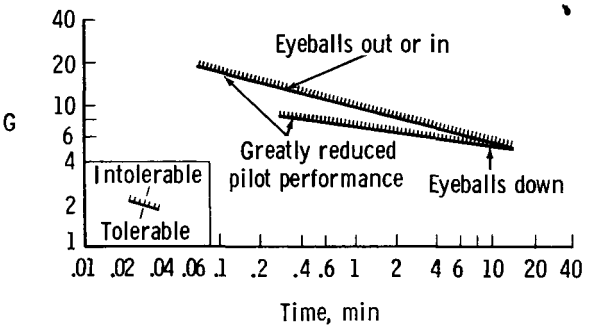


FIGURE 27.—*Time tolerance to acceleration boundaries.* (Smedal et al.,[154] 1960.)

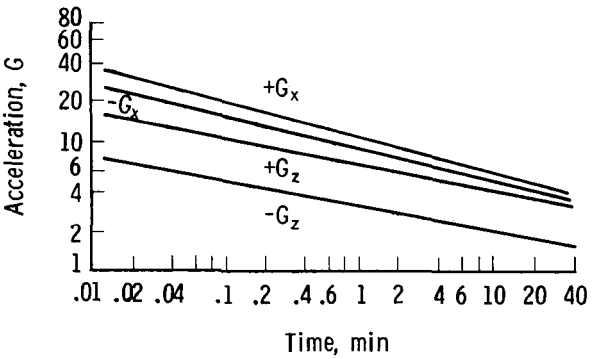


FIGURE 28.—*Average acceleration tolerances for transverse supine acceleration ($+G_x$), transverse prone acceleration ($-G_x$), positive acceleration ($+G_z$), and negative acceleration ($-G_z$).* (Chambers,[35] 1963.)

tolerable in the $\pm G_x$ vector, as has been well demonstrated by the astronauts. Glenn, for example, was exposed to a peak of $+7.7G_x$ during launch, Carpenter to $+7.8G_x$, and Schirra to $+8.1G_x$.

While a diagram of the type discussed is useful for establishing broad limits, it does not fully express the situation. Since it records only plateau acceleration, it ignores the G-time consumed in attaining the plateau and receding from it—although the latter of course takes place after a tolerance threshold has been established. However, in the higher plateaus, depending on the rate of onset, a significant G-time may be involved in reaching the level required, and thus, in terms of total impulse, the threshold is proportionately greater than at lower levels.

To overcome this problem, Beckman et al.[15] (1953) used a different technique of analysis. They exposed their subjects to levels of +6, 8, 10, 12, and 15G_z at rates of onset of 3.5, 7.1, 8, 9.6, and 8G per second respectively. The time to attain a given plateau, the duration at plateau G to unconsciousness, and the duration to offset were plotted. The curves were analyzed as to the total time the subjects were exposed to accelerative loads greater than 3, 4, and 5G. The graph, figure 29, shows that the duration at 6G before unconsciousness is greater than the duration at higher levels. The areas under the preplateau load-duration curves for 8G and higher, however, are not significantly different statistically. Thus, increase in the magnitude from 8 to 15G_z does not significantly alter the duration of stress required to produce unconsciousness. At the 6G level, however, the times during which the loads are greater than 3, 4, and 5G are considerably longer than those for 8G and higher.

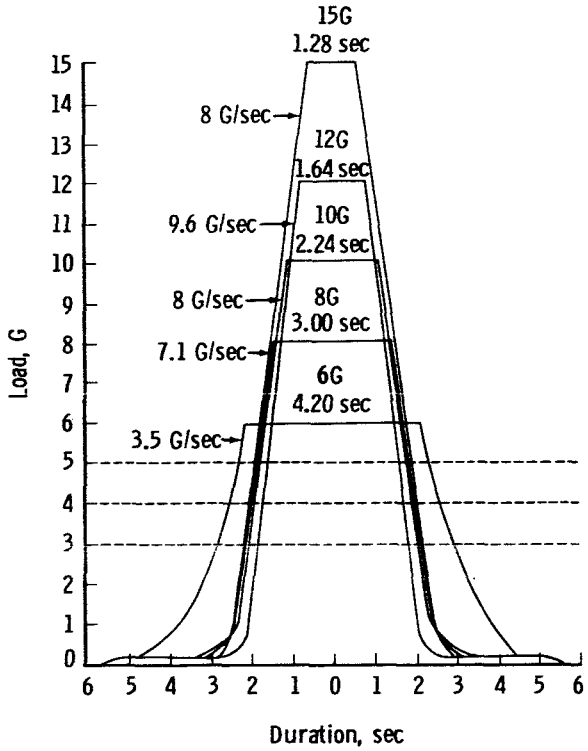


FIGURE 29.—*Load curves of experiments which produced unconsciousness, constructed from mean values.* (Beckman et al.,[15] 1953.)

RATE OF ONSET

From the physiological viewpoint, as has been noted, there is a latent period, particularly when acceleration is applied in the G_z vector, before the body can muster its compensatory resources. Thus, at least to 8G_z as noted by Beckman et al.,[15] tolerance is reduced with higher rates of onset. The precise relationship has been very little investigated.

Stoll [169] (1956) analyzed the records of 40 experiments on 15 subjects in terms of rise time, time at maximum acceleration (+G_z), level of maximum acceleration, and rate of onset. Figure 30 illustrates the duration intensity curve obtained, with the rates of onset superimposed. The difference between the heavy solid line (observed end-points) and the heavy dashed line (extrapolated values) represents the threshold gain obtained by compensatory measures. The end-points produced by different rates of onset are shown by the small arrows. From these data a nomogram was developed (fig. 31) which, although empirical, indicates the expected duration befor grayout at a given plateau and rate of onset.

Beckman quotes Jasper's work with electroretinograms in which he concluded that with high rates of onset the central portion of the visual pathway was interrupted first, whereas with lower rates of onset the retinal portion was interrupted first. Maciolek [122] (1955) similarly reported that the mean blackout threshold in seven subjects under acceleration with rate of onset of 1G per second was 3.7G, while that with rate of onset of 0.7 to 0.1G was 6.2G. This statement that tolerance is reduced with higher rates of onset is in direct contradiction to the statement by Bondurant et al.[19] (1958) that slower rates of onset shorten tolerance time. It may be that there is an optimum rate or rates

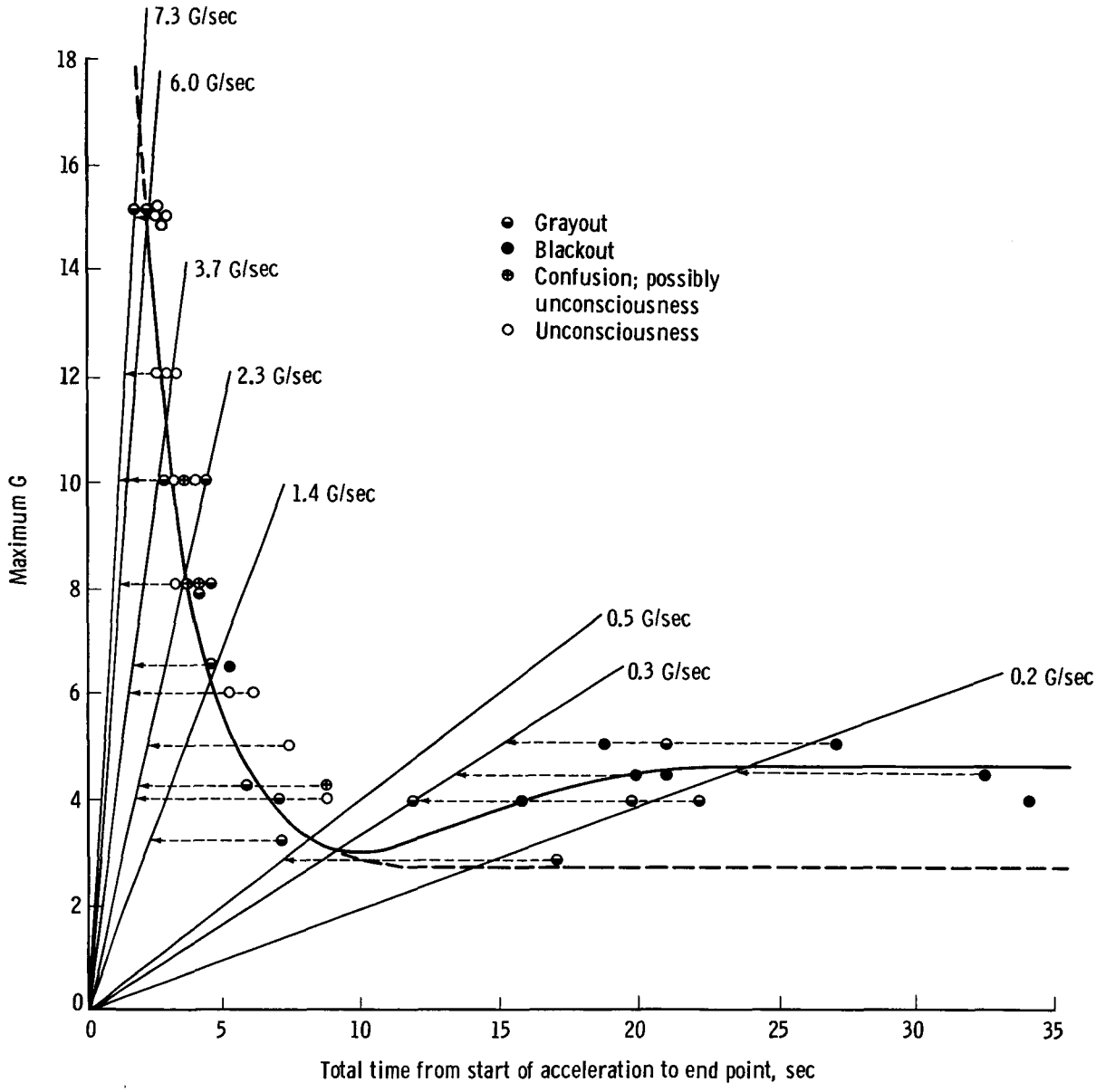


FIGURE 30.— *G-tolerance curve with various acceleration rates.* (Stoll,[169] 1956.)

where the disadvantages of increasing the total impulse by reducing the rate of onset and prolonging the duration are balanced by providing time to allow maximum compensatory response. There is room for definitive study on the effects of varying rates of onset.

POSTURE

The question of posture within a given vector has been examined by several investigators. The effect of altering the head angle (Steiner and Mueller,[164] 1961) has been previously noted, and there are considerable effects to be observed by altering the posture within the $\pm G_z$ vector. Most of the work has been done in establishing the optimum position in the $+G_x$ vector.

Starting with subjects exposed to $+G_x$ in the fully supine position, Ballinger[7] (1952) showed that a limit of chest pain and dyspnea

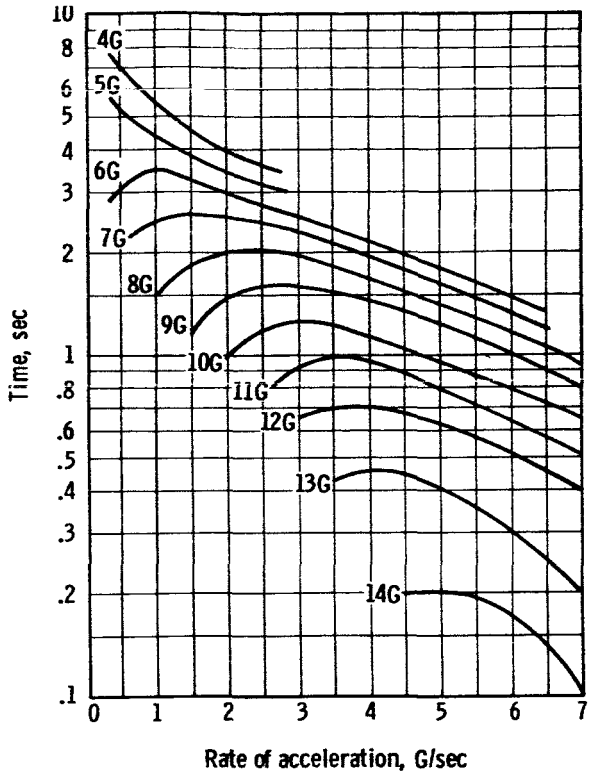


FIGURE 31.—*Nomogram relating acceleration rate to time to grayout.* (Stoll,[169] 1956.)

was reached at $8G_x$. A better position was established by elevating the chest and head and raising the knees to head level with the legs outstretched in a reclining seat.

Bondurant et al.[19] (1958) experimented with various positions in the forward acceleration vector, with the results shown in figure 32. The arrows in this and the subsequent figure refer to direction of acceleration, not of inertial forces. Figure 33 illustrates the advantages and disadvantages of a variety of positions. Tolerance in the conventional seated position (B2) is limited at 8G by dyspnea and chest pain. In addition there is a component of negative G from the backward tilt of the trunk. If the angle of the trunk relative to the direction of acceleration is greater than 70°, a severe quasi-pleuritic anterior chest pain limits tolerance at about 7G. As the angle is decreased below 70° (B3) there is more longitudinal application of the inertial force, and blackout limits the tolerance at progressively lower levels. The best tolerances are found with the subject inclined in the direction of acceleration at a 65° to 70° angle. Blackout may occur in positions B and D, although position B has a higher threshold. The chief limiting factor in these positions is of course dyspnea. Asymptomatic petechiae also tend to occur.

The ideal position, then, for forward acceleration appears to be a seated posture, forward facing, with a 20° inclination of the trunk in the direction of acceleration, hips flexed to bring knees to eye level, and lower legs extended.

The optimal backward position is illustrated in position E. An effective negative G component is introduced, however, if the head and trunk move forward (E1), with a reduction in tolerance dependent on the angle. If the legs are extended (E2), intense calf and thigh pain limit tolerance to about 5G.

TOLERANCE TO TUMBLING AND COMBINED ACCELERATIONS

Very little is known about the upper limits of human tolerance to simple tumbling or tumbling in a decelerative field. Most of the studies have involved animals. Tolerance to oscillation, as represented by vibration, will not be considered here.

Edelberg[63] (1961) has prepared diagrams, shown in figure 34, which indicate the approximate limits as currently accepted. It is probable, however, that these limits err on the low side, and that work with an epicyclic centrifuge would provide more definitive data and show an increased threshold.

Chambers[34] (1961) has produced a table derived from the personal opinions of pilots exposed to piloting tasks within combined G fields, obtained by adding angular acceleration from the motion and positions of two gimbals on the Johnsville centrifuge to the radial acceleration of the centrifuge arm. The fields are ranked in increasing order of difficulty, the first being the easiest and the

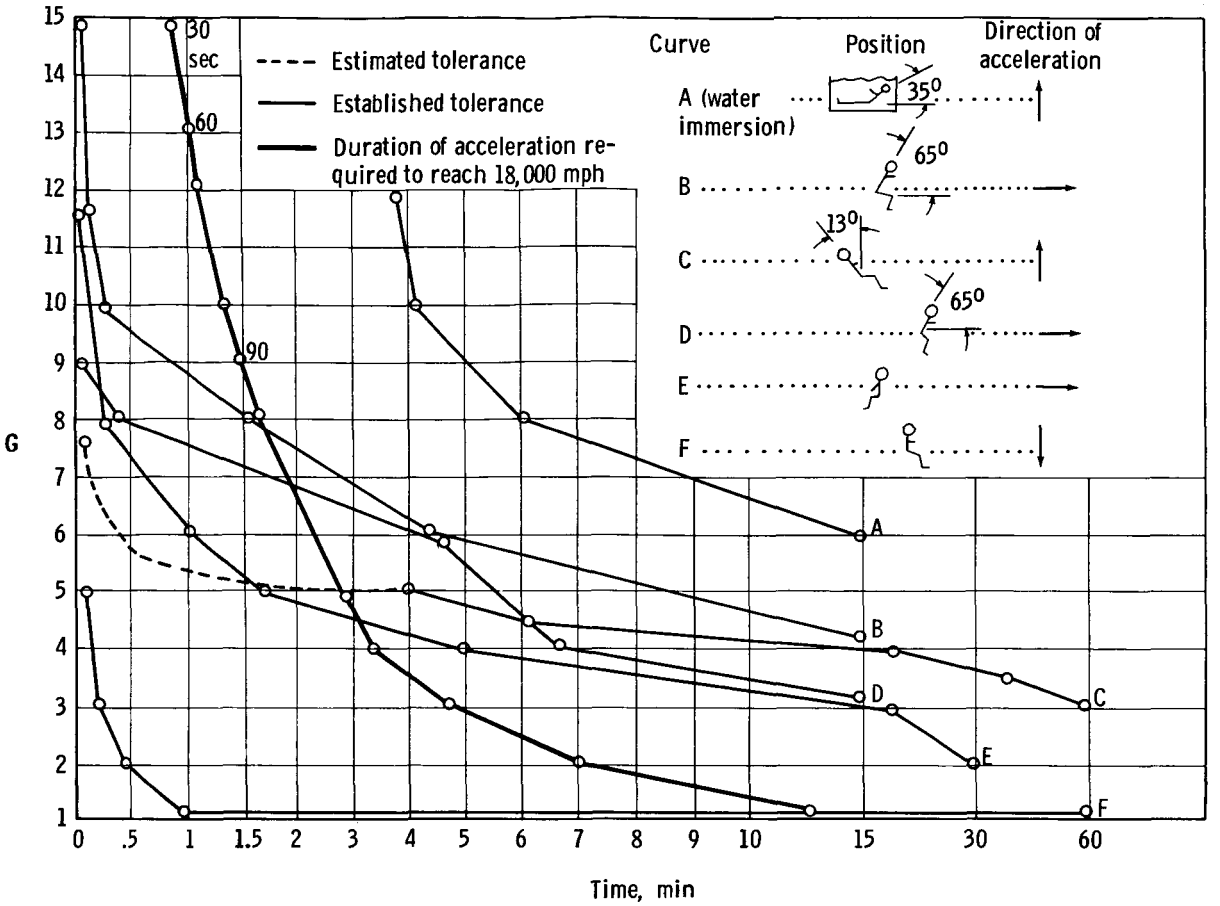


FIGURE 32.—*Effect of position on tolerance to acceleration.* (Bondurant et al.,[19] 1958.)

last the most difficult. It will be noted that the table is presented in terms of acceleration fields, a_x and a_z, and not in terms of inertial resultant.

a_x		a_z
0	by	+1
−3	by	0
0	by	+4
−4	by	0
+5	by	0
−5	by	0
−6	by	0
+6	by	0
0	by	+5

The exposure time for most of these runs was 2½ minutes, although some exposures were as short as 2 minutes and some as long as 7 minutes. The pilots were fully conscious in all cases.

Unfortunately, pilot opinions on combinations of $\pm a_x$ with $\pm a_z$ are not quoted in the table, although the author states that in the $+a_x$ by $+a_z$ acceleration field tingling in the toes and feet was observed, sometimes accompanied by pain, and a feeling of stiffness occurred in the lower extremities. There was also a slight blurring of vision.

Further studies in tolerance to combined G fields would be of value.

SPACE FLIGHT SIMULATION

While it is essential to establish academic parameters of acceleration tolerance in terms of load, onset, and time, it is equally necessary to determine whether an individual can withstand the irregular profile of launch and reentry acceleration.

As noted earlier, a specific impulse of 800 G-seconds is required to attain orbital veloc-

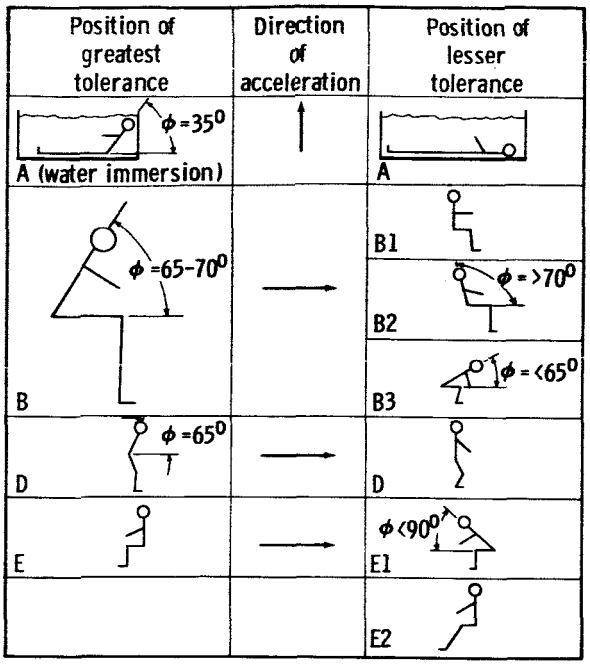


FIGURE 33.—*Variations in position which decrease tolerance to acceleration.* (Bondurant et al.,[19] 1958.)

ity. This of course has to be established over a series of peaks which subjectively approximate a series of parabolic increases with time, separated by discontinuous decreases.

The effect of acceleration imposed in this manner can be examined by using a profile of theoretical repetitive peaks as illustrated in figure 35. The response to profiles of this type was studied by Preston-Thomas et al.[138] (1955), who showed that three and four peaks with a maximum of 8 and 6G, respectively, from a baseline of 2G could be tolerated when applied over a duration of 350 to 450 seconds. Similarly, Bondurant et al.[19] (1958) showed that three peaks with varying rates of onset could be tolerated with little difficulty.

In each of these cases, however, the situation is unrealistic since the use of fuel during acceleration will change the magnitude of the accelerated mass and vary the rate of onset. A more realistic profile is illustrated in figure 36. Chambers[34] (1961) showed that a profile of this type was tolerable. The effects of this type of acceleration will be discussed further in connection with performance capacity.

TOLERANCE TO VERY PROLONGED ACCELERATION

Several investigators have studied the effects of acceleration of +3G and +4G sustained for up to 1 hour (for example, Bondurant et al.,[19] 1958, and Miller et al.,[128] 1959), but there are few instances on record of exposure of man beyond 1 hour. Clark[40] (1960), however, described an investigation with a subject who rode the Johnsville centrifuge for 24 hours at 2G unrestrained in a reclining seat. This was tolerated, although symptoms and discomfort developed in the course of the run. These included rotational illusions, nausea, and Coriolis type reactions on head movement, headache, and abdominal discomfort. Although the subject could walk with little difficulty early in the experiment, a similar attempt 22 hours after the start produced dimming of the peripheral vision. Another interesting phenomenon was a rise in the white blood count from 11,300 to 22,000 per cubic millimeter. The cause of this is obscure. Sixteen hours after the start anesthesia developed in the ring and little fingers of the left hand, and some tingling remained for about 2 months after the experiment. This was presumably a pressure neuritis of the ulnar nerve, no doubt from the arm rest. A feeling of lightness lasted for about 30 minutes after cessation of acceleration, and abrupt head movement during this time could still cause nausea and retching.

With the eventual engineering possibility of application of acceleration for prolonged periods, more studies are required of the effects of 2 and 3G for periods greater than 1 hour.

Comment may be made here on work by Hamilton Smith's group at the University of California at Davis (Burton et al.[31] 1963). Although this work was undertaken on chickens and does not have direct application to man, it is interesting to observe that during nearly continuous exposure to 3G for 2½ months (a major portion of the growing period of a chicken) several patterns were isolated:

(1) Peracute—Death occurring within 12 hours of onset of symptoms.

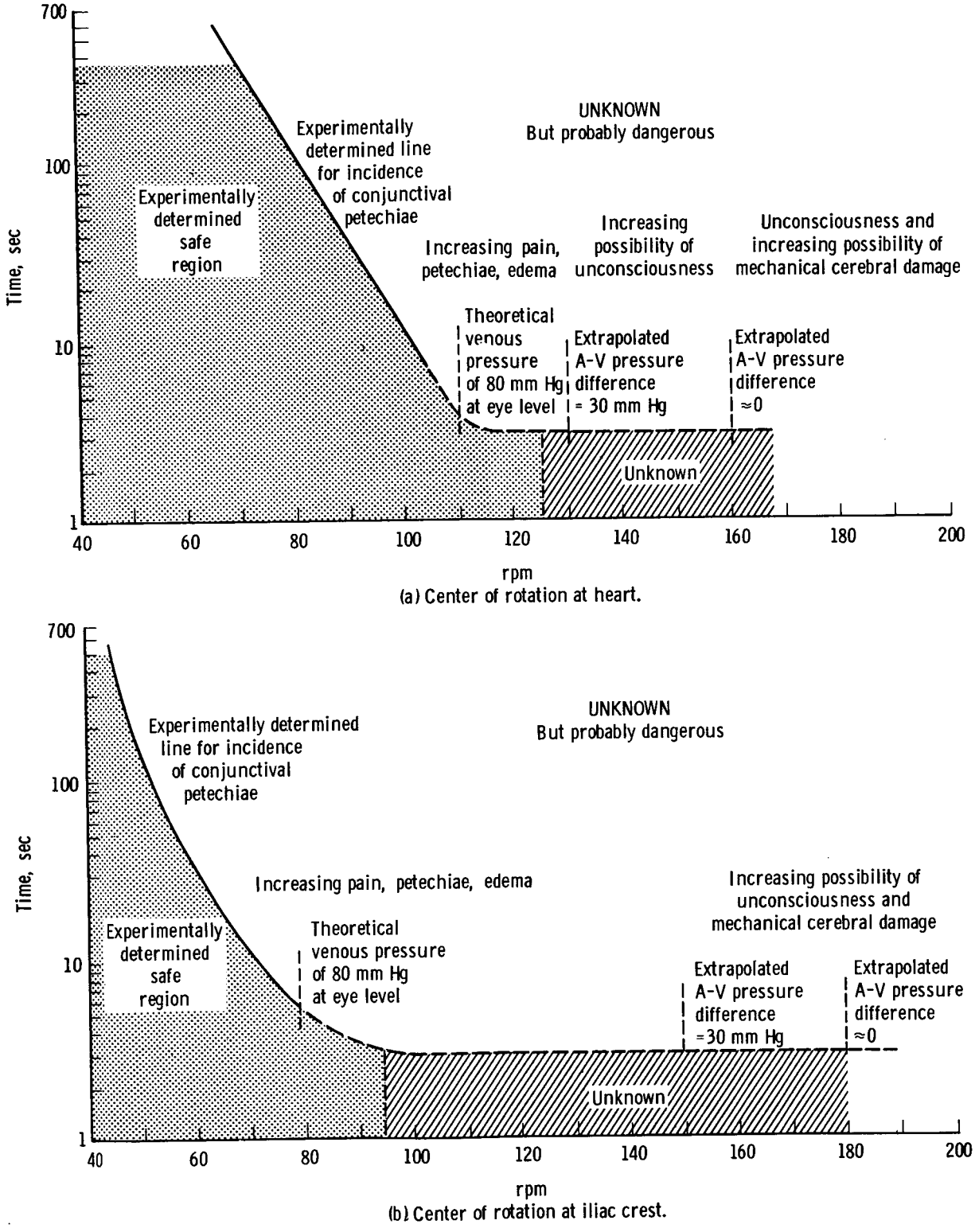


FIGURE 34.—*Human tolerance to simple tumbling (no superimposed deceleration). (Edelberg,[63] 1961.)*

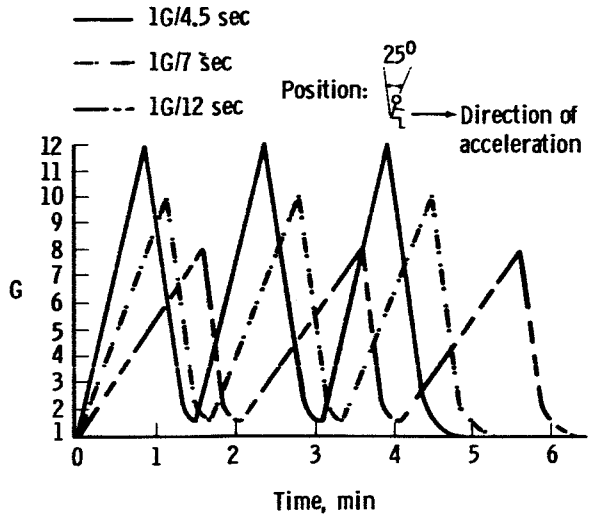


FIGURE 35.—*Some three-stage rocket acceleration patterns that are sufficient to exceed orbital velocity and are tolerable to man.* (Preston-Thomas et al.,[138] 1955.)

(2) Acute—Progressive development of symptoms, and death within 24 hours. Death in both cases was associated with hemorrhage in one form or another.

(3) Subacute—Early development of symptoms, followed by remission and improvement and eventual death.

(4) Recovery—Early development of symptoms, followed by progressive reversal and return to normal within 24 hours.

(5) Asymptomatic—No observable symptoms.

The birds included an unselected strain and a strain selected as the sixth generation surviving chronic acceleration. A greater number of the latter survived the experience. Sex differences were also noted. Thus, 59% of selected males and 69% of selected females survived, whereas only 32% of unselected males and 16% of unselected females survived.

This group has plans for exposure of man to very prolonged acceleration (3 to 12 months) which bear watching.

RESTRAINT

The standard seat harness, normal in military aircraft, is inadequate for protection under sustained acceleration applied in the $+G_x$ vector, and still more so in the $-G_x$. To counter this problem, Clarke et al.[47] (1958) carried out tests in the $\pm G_x$ vector with an integrated helmet and restraint suit which bound the subject closely to a fighter aircraft seat. Good tolerances were established in both vectors.

Meanwhile, using a different approach, Clark et al.[44] (1959) tested custom-molded contour couches which provided inclination of head, trunk, thighs, and legs, optimal both for acceleration tolerance and useful performance.

These couches, while providing excellent support in the $+G_x$ vector, were inadequate in the $-G_x$ vector. Consequently, Smedal et al.[156] (1960) tested a restraint system, which came to be known as the Ames system, that included a posterior molded couch, a restraint helmet and supporting face and chin pieces secured into the mold, a chest and pelvic torso support, and nylon netting supports for upper arm, thigh, and lower leg. In addition, subjects wore a G-suit. This system, while cumbersome and not completely satisfactory under $-G_x$, was found to give the best restraint yet devised.

There is, however, a need for a universally adjustable contour couch with adequate restraint, yet freedom for action, applicable to wide ranges of body type and vehicle. Chambers[35] (1963) reports that such a couch is under development.

The Air Force Flight Dynamics Laboratory (Peterson,[131] 1964) has been testing the possibilities of developing seats with raschel nylon net as the primary back, seat, and leg

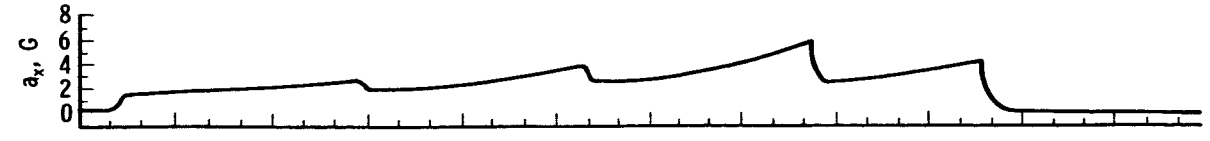


FIGURE 36.—*Typical time history of acceleration for four-stage launch simulations.* (Chambers,[34] 1961.)

support surfaces. The objectives are:

1. Maximum comfort for long periods during all phases of the mission.
2. Maximum support and restraint during the sustained acceleration phases of the mission.
3. Adequate support during periods of low-frequency high-amplitude vibration.
4. An integral total-body restraint system.
5. Sufficient adjustments, including angular adjustability, to accommodate the 5th through the 95th percentile crew member.
6. Accommodation of pressure suits as well as regular flying suits.
7. Ultimate provision of an integrated arm-restraint device and a three-axis hand control.
8. Ultimate provision of an emergency encapsulation device.
9. Lightness, easy maintenance, durability, and crew appeal.

Several seat systems using raschel knit cloth were designed and evaluated, and the technique was found to provide excellent body support during extended acceleration up to $+16.5G_x$, semisupine. Under severe vibration and impact acceleration, however, undesirable rebound was encountered. The investigators believe this can be overcome, and are continuing development. It is considered that this technique holds promise.

Consideration is currently being given to the use of a parachute-harness type of restraint for occupants of the Lunar Excursion Module. Although the expected sustained acceleration (as opposed to impact) is relatively low, it would be advisable to check the ability of the harness to provide adequate restraint.

PROTECTION AGAINST ACCELERATION

Tolerance of acceleration may be improved under some circumstances by the use of protective measures and devices. Straining and grunting in a tight turn was early practiced by pilots to improve their tolerance, and culminated in the development of the M–1 maneuver, namely, tensing of muscles and straining, with forced expiration through a partially closed glottis. Changing of posture to meet acceleration has also been practiced, notably in World War II, where hunching into the cockpit was found to improve the tolerance of pilots in high-acceleration maneuvers.

The history of protective devices goes back to the work of the French Air Force in World War I. Aldworth, in a personal communication to Wood et al.[191] (1963), described their use of a device consisting of a water-filled bladder on which the pilot was seated. This cushion was connected with bladders over the thighs and abdomen. Increase in body weight under $+G_z$ acceleration applied pressure to these bladders. The outcome of this work is not known.

Poppen [137] (1932) later devised an abdominal belt which could be inflated prior to an accelerative episode. Later, as reported by Gauer [78] (1950), the Germans developed a water-filled "jacket" which provided counterpressure to the body, proportional to the increase in hydrostatic pressure. In reality, the "jacket" was a pair of trousers. Using the same principle, Franks and his colleagues at Toronto, as reported by Davidson et al.[55] (1954), developed in 1939 the first operational anti-G suit, which was utilized with success in Western Desert air fighting. Water-filling was cumbersome, however, and meanwhile Cotton in Australia had developed a suit which utilized suitably placed air bladders instead of water. Refinement of the suit design, and the valve which metered the air into the bladder system led to the development of the current G–3A and G–4B suits still sometimes used in interceptor aircraft. Still further modification led to the wrap-around CSU 3/P cutaway type of anti-G suit which improves blackout tolerance in the $+G_z$ vector by about 2G (Leverett et al.,[112] 1961).

The physiological effects of anti-G suits and similar devices, which were originally defined at Toronto and the Mayo Clinic, are summarized by Wood and Lambert [189] (1952). It was shown that inflation of a suit at 1G produces an initial rapid rise in arterial pressure. This is followed almost immediately by an abrupt decrease in the heart rate,

probably due to a depressor reflex originating principally in the carotid and aortic areas, although other mechanisms in the thorax and abdomen may be involved. Cardiac irregularities may occur. The same pattern is seen when the suit is inflated under acceleration at $+5.5G_z$. With inflation, the arterial pressure rises above normal value at heart level but remains below the normal value at head level. Since the pressure in the carotid sinus is not elevated, slowing of the heart rate does not occur.

Analysis of contributions of portions of the suit shows that the whole suit gives protection varying from 1 to 2G, in terms of blackout threshold, while leg bladders alone give protection of about 0.2G. Abdominal bladders without leg bladders protect to about 1.2G, while arm cuffs alone produce a hardly measurable protection. Arm cuffs added to a full suit will raise the protection by a further half G.

In a later paper, Lindberg et al.[116] (1960) point out that the decrease in cardiac output that occurs under applied G_z acceleration is not significantly altered by the use of an inflated anti-G suit; hence, the protection afforded by the suit is not associated with a relative increase in cardiac output. They observed, however, that inflation of an anti-G suit under G_z acceleration is associated with a relative increase in mean aortic pressure which acts to counteract the increased hydrostatic pressure. They consider that the suit acts by increasing the systemic vascular resistance, as well as reducing peripheral pooling.

While the effect of an anti-G suit in the G_x vector would not be expected to be so dramatic, Clark et al.[44] (1959) found that it made runs below $15G_x$ more comfortable and reduced the visual effects in subjects unpracticed in straining techniques. Above 15G, the suit was of no benefit in increasing G tolerance, and, in fact, was found to make straining more uncomfortable. A water-filled immersion suit, however, prevented the occurrence of petechiae, which raises again the possibility of utilizing water suits for G_x protection.

The possibilities of subtotal immersion were originally investigated by Wood et al.[190] (1946), who used a shoe-shaped tank in which the subject was submerged to the level of the third rib in the seated position, holding his breath. Gray and Webb[87, 88] (1960, 1961) did similar work, using initially the same tank. A level of $16G_z$ was attained. Subsequently a G-capsule was constructed in which the subject was completely submerged, breathing ambient air through a mask at 1G, and air pressurized above hydrostatic at higher acceleration. The pressurization for respiration was limited at the $25G_z$ level because of ear and sinus pain, but during breath-holding experiments the subject reached a level of 26 to $31G_z$ for a 5-second duration. Minor damage, however, occurred in the sinuses. All subjects also experienced abdominal pain, which could be eliminated by tightening the abdominal muscles. A great advantage of total water immersion is the ability under high acceleration to move the limbs freely. This technique has great promise but is currently severely limited in practical use because of the extreme weight penalty.

While water submersion provides a great increase in the tolerance, it does not prevent the hydrostatic shifts occurring within the chest cavity, namely, pulmonary congestion posteriorly and overexpansion anteriorly.

This problem has been attacked in an interesting manner by Black-Schaffer et al.[17] (1961) and Black-Schaffer[16] (1962). Using a modification of Newton's Second Law, as follows:

$$F = V(d_1 - d_2)a$$

where d_1 and d_2 are the specific gravities of an immersed homogeneous body and the immersion fluid, V is the volume of the body, and a is acceleration, they argue that when d_1 equals d_2 the force exerted within the system is zero. The significant word here is "homogeneous."

On this basis, the authors showed in the first paper that deep hypothermia combined with immersion in a sodium chloride solution of approximately the same density can pro-

tect adult mice against a load of 1,800G for 15 minutes. The protection, however, was not complete. One animal survived for 15 months, but the others died, mainly because of obstruction of the small caliber nasal passage by blood and mucus on recovery from acceleration.

In the second paper (Black-Schaffer,[16] 1962) the author extended the technique to hamsters, which were able to survive indefinitely after a load of 2,200G for 30 minutes. Rats exposed to the same load failed to survive more than 5 minutes.

It should be noted that results of exposure of small animals to high levels of acceleration have limited applicability to large animals because of great differences in relative chest dimensions. The effects of acceleration on the lungs are directly related to the superior-dependent dimension exposed to acceleration (E. H. Wood, personal communication).

While it is not suggested that this technique can be applied to man at this time, it has obvious possibilities which should be further explored.

ADAPTATION TO ACCELERATION

Adaptation to the effects of acceleration can occur with habituation. Franks [70] (1960) states that during flying training, tolerance to G_z will improve by half a G.

The effect of conditioning by repetitive exposure at very short intervals was examined by Hallenbeck [89] (1945), who exposed subjects to six 10-second episodes of 4.2G at increasing intervals of 4.7, 9.6, 19.4, and 29.1 seconds. Improvement in blackout tolerance was observed when the interval between exposures was 10 seconds or less.

It is of interest that Russian cosmonauts, as detailed by Isakov and Stasevich,[100] 1964, are exposed to a program of graduated physical training, including regular experience on lightweight swing-type centrifuge, with the object of creating new conditioned reflex patterns governing circulatory compensation. An increase in tolerance of 1 to 2G is claimed by users of this technique.

Long-term conditioning is known in human subjects, although it does not appear to have been carefully analyzed. In animals, Frazer et al.[73] (1958) observed adaptation in the response of rats to a load of 19.2G, when one group had been conditioned to 2G, 5 days a week for 6 weeks, and another to 12G, 5 days a week for 6 weeks. The latter group had a significantly longer survival time than a control (unconditioned) group, while the former had a shorter survival time. The authors give no explanation of this latter finding, which has not been previously noted. At this time, only speculations are possible.

PHARMACOLOGICAL METHODS

Numerous drugs and substances have been tested to determine their effect on acceleration tolerance.

Gauer [78] (1950) details some of his wartime work when he tested the effects of caffeine, Pervitin, atropine, oxygen, and carbon dioxide. The three drugs and oxygen were shown to be ineffective, but inhalation of 6% carbon dioxide resulted in an increase of approximately 10% in $+G_z$ tolerance, no doubt because of the peripheral vasoconstriction produced and because of dilation of the CNS arterial circulation.

In another approach, Polis [135] (1961) investigated substances to stimulate pharmacologically the effect of hypophysectomy, and found that Lucidril, the dimethylaminoethyl ester of parachlorophenoxacetic acid, significantly increased the tolerance of rats to 20G. The median survival time was almost three times the control survival time (table 17). The effectiveness of the drug persisted for a period of 4 hours after injection, but a latent period of 3 to 4 daily injections seemed necessary before the increase in tolerance became evident. The activity of the drug also appeared to be dependent on the dose, with no changes at 50 mg, significant increases at 75 mg, and still greater increases at 100 mg. Polis quotes the work of French pharmacologists to indicate that the action of the drug is probably mediated via the hypothalamus, although he points out that its relationship to acetylcholine requires investigation. This is a new approach to the pharmacology of acceleration tolerance and should be explored.

TABLE 17.—*Experimental data for the effect of Lucidril on the rat's tolerance to acceleration.* [Polis,[135] 1961.]

No.	Type of treatment	Conc. of drug, %	Amount of drug injected intraperitoneally		No. of injections	Average weight of rats, gm	No. in group	Survival time of population, min			Median time difference between control and treated animals, min	Sig. of diff. at 5% level	Notes
			ml	mg				84%	50%	16%			
A	Control					235	19	5.5	12.5	27.6			
1	Lucidril	5	0.3–0.5	15–25	6	207	17	15.5	33.3	70.8	+20.8	Yes	
B	Control					267	35	5.8	13.0	29.0			
2	Lucidril	5–10	0.3–0.5	15–50	4–6	256	55	9.2	20.9	47.9	+7.9	Yes	
3	Lucidril	5–10	0.3–0.5	15–50	4–6	256	40	11.3	24.0	52.0	+11	Yes	Spun within 3 hr of last injection.
4	Lucidril	10	0.5	50	3–4	278	27	9.0	20.2	46.0	+7.2	Yes	
5	Lucidril	5–10	0.3–0.5	15–50	4–6	256	15	5.8	12.1	22.4	−0.9	No	Spun 4–8 hr after last injection.
6	Lucidril	10	0.5	50	1	273	35	5.9	10.4	19.5	−2.6	No	
7	Lucidril	5	0.3	15	5	282	12	9.3	21.0	49.0	+8.0	Yes	Significantly different from series 6.
C	Control					249	20	3.5	10.2	30.5			
8	Lucidril	10	0.3	30	6	238	20	6.2	14.5	34.0	+4.5	No	Lucidril in acid form.

CHAPTER 5

Performance During Sustained Acceleration

Satisfactory performance demands adequate perception of appropriate stimuli, integration and correlation of these stimuli with previously established patterns, and coordinated effector action. Thus there are many ways in which exposure to acceleration may interfere with a pilot's performance. Perception may be disrupted by interference with the sensory process; integration and correlation may be impaired by disturbance of cerebral oxygenation; and effector action may be opposed by the forces developed.

Of primary importance are the visual, vestibular, kinesthetic, and auditory senses.

VISION

The subject of vision under imposed acceleration has received a great deal of attention because of its importance in flying activities and the dramatic effects of, in particular, $+G_z$ acceleration. A comprehensive review of the subject was made by W. J. White and Monty [183] (1963), from which much of the following is obtained.

Visual Threshold

Grayout and blackout are of course the most striking effects of acceleration and it is probable that they represent readily distinguishable points on a continuum of diminishing perception. Cochran et al.[49] (1954) established thresholds on 1,000 subjects exposed to $+G_z$ acceleration (table 18).

TABLE 18.—*Thresholds for $+G_z$ acceleration.* [Cochran et al.,[49] 1954.]

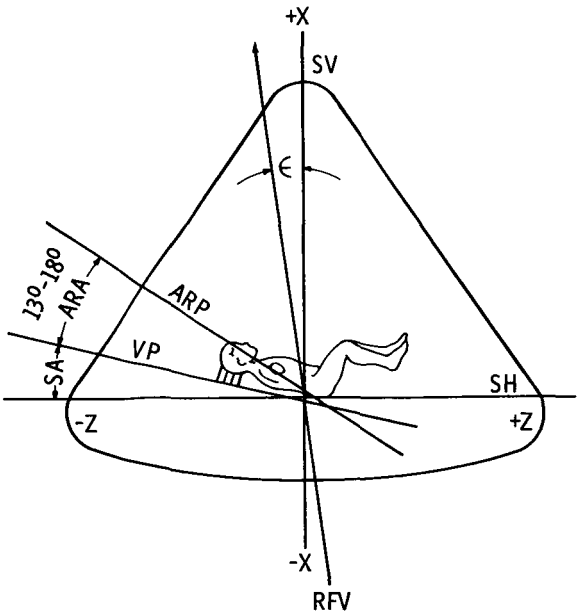
Criterion	Mean threshold, G	Standard deviation, G	Range, G
Loss of peripheral vision	4.1	± 0.7	2.2–7.1
Blackout	4.7	± 0.8	2.7–7.8
Unconsciousness	5.4	± 0.9	3.0–8.4

With regard to transverse acceleration, Chambers [34] (1961), as has previously been noted, found some loss of peripheral vision at $+6G_x$, increasing to marked loss at $+12G_x$, and recurrent blackout at $+15G_x$. The physiology of this phenomenon has been discussed, and it was shown how the onset of grayout and blackout were related to changes in hydrostatic pressure.

Alexander (personal communication), in an unpublished work on behalf of NASA, determined what he defines with respect to the onset of grayout as the "effective physiological angle" (EPA). The terminology is illustrated in figure 37. Thus, the EPA is the sum of the seat angle, the angle ϵ, and the aortic retinal angle. The seat angle is the angle between the seat back and the horizontal; angle ϵ is the angle between the spacecraft vertical and the resultant force vector of the accelerating spacecraft. The aortic retinal angle is obtained by direct measurement on a lateral thoracic radiograph of the angle between a perpendicular through the aortic arch and a line joining the aortic arch with the outer bony canthus of the eye. This angle was found to range from 13° to 18° with a mean of approximately 15°.

Grayout thresholds were obtained for different effective physiological angles, and a curve was prepared as illustrated in figure 38. Thus, in the current Apollo position, with a 15° aortic retinal angle, 2° back angle, and value of 6.5° for ϵ, the effective physiological angle will be 23.5°, and grayout will be expected at about $7\frac{1}{2}G_x$.

There would appear to be a continual change in absolute threshold of vision under acceleration, that is, the minimum light intensity at which a stimulus can be perceived. White [183] (1960) studied these changes and



ARA = Aortic retinal angle
ARP = Aortic retinal plane
∈ = Angle inscribed by RFV with SV
RFV = Resultant force vector
SH = Spacecraft horizontal
SV = Spacecraft vertical
VP = Vertebral plane
SA = Seat angle
EPA = Effective physiological angle, SA+ARA+ ∈

FIGURE 37.—*Grayout terminology.* (Alexander, unpublished, 1964.)

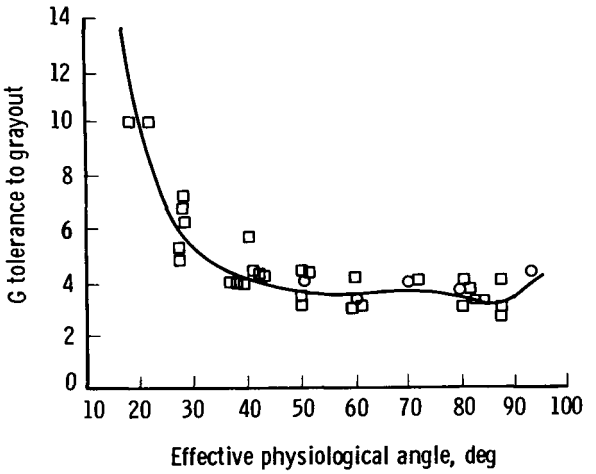


FIGURE 38.—*Physiological tolerance.* (Alexander, unpublished, 1964.)

showed that at +3G the foveal threshold was almost double that at 1G, and at +4G, it was 3.4 times that at 1G, when measured at the 50% probability level. In the periphery the luminance of the stimulus has to be increased 1.5 times at 2G, 3 times at 3G, and 4 times at 4G. Similarly, a decrease in differential threshold, the minimum perceptible difference between a pair of stimuli, is observed, most marked with positive acceleration and for low background luminance.

Chambers [35] (1963) quotes other studies in which visual brightness discrimination was examined under four levels of background luminance, four levels of positive ($+G_z$) acceleration, and five levels of transverse ($+G_x$). For each of the four $+G_z$ conditions (1, 2, 3, and 5G) the visual contrast requirements increased as the background luminance decreased, and for any given background luminance the higher acceleration levels required more brightness contrast. Similar results were shown for the $+G_x$ exposures (1, 2, 3, 5, and 7G). The G_z acceleration consistently imposed higher contrast requirements than did the G_x.

The effect of oxygen on vision during acceleration was reported by Chambers and Hitchcock [36] (1963). Their work indicated that during accelerations of $+3G_z$, $+4G_z$, and $+5G_z$ subjects breathing 100% oxygen required less contrast to maintain a target at the minimally discriminable brightness level against a diffuse background luminance of 0.03 foot-lambert. Similar results were found in the $+G_x$ vector. Subjects breathing normal air at a positive pressure required increasing amounts of contrast for discrimination as the acceleration increased, while those breathing 100% oxygen at positive pressure showed results similar to those breathing 100% oxygen at ambient pressure. Thus the significant variable would appear to be the oxygen.

Visual Acuity

Visual acuity, which is a foveal function, also decreases linearly with increase in acceleration. This decrease, however, is independent of body position, as was shown by

W. J. White and Jorve [184] (1956), and consequently is independent of change in hydrostatic pressure. The authors suggest that this loss of acuity is related to displacement of the lens in the direction of the acceleration vector. It is believed, however, that they are really referring to the inertial vector. Frankenhaeuser [69] (1958) also found a loss in visual acuity at 3G corresponding to 16%.

In the $-G_x$ vector, visual disturbances become marked at $-6G_x$ to $-8G_x$. For some time there was controversy as to the cause, particularly with respect to the possibility of corneal distortion. Using an ingenious optical technique, Smedal et al.[155] (1963) reflected the image of a placido disk on the cornea and photographed it under acceleration. They found no distortion attributable to corneal deformation. Intermittent watering of the eyes, however, distorted the reflection above $-6G_x$.

Visual Fields

Very little work has been done in determining the degree of narrowing of the visual field that occurs with acceleration. White and Monty [185] (1963) quote Hallenback as stating that at $+4.4G_z$ (range of $+3G_z$ to $+6.5G_z$) the field is narrowed to an arc of less than 46°.

Eye Movements

The limitation in ocular motility observed by Beckman et al.[14] (1961) has been previously noted. There is no indication, however, whether eye movements prior to the occurrence of LOMA are in any way reduced.

Pupillary Reactions

Beckman et al.[14] noted that pupillary dilatation began with loss of peripheral vision. This is in contrast to the statement by Wilson and Canfield [188] (1951), quoted by White and Monty,[185] that the pupil dilates immediately on exposure to acceleration. Smedal et al.[155] (1963) used a photometer mounted in the centrifuge cabin and determined that accommodation ability was unaffected by acceleration.

Reading Tasks

Reading tasks of course demand an intact performance loop and are more than measures of vision. Warrick and Lund [173] (1946) compared the results of dial reading in 34 pilots at 3G and 1½G. At 3G, 24% of readings were erroneous, as compared with 18% at 1½G. This of course is a clear indication of significant performance decrement prior to grayout level. The response, however, varies with the luminance, as we have seen. White and Riley [186] (1958) varied the luminance in a dial-reading test from 0.004 millilambert to 42 millilamberts and found that with exposure to 1, 2, 3, and $4G_z$, there was no difference at the highest level of illuminance under all four conditions. With lowered illuminance there was increase in error with increase in acceleration above the $2G_z$ level. These figures illustrate the importance of providing good instrument lighting under acceleration.

Reaction Time

Reaction time, too, is more than a test of visual adequacy, but will be considered at this time. Burmeister, as quoted by Ham [90] (1943), found increased reaction time to a light signal in 1939, although surprisingly Franks et al.[72] (1945), in their wartime work, found no increase with 7853 stimuli during 626 tests at $+2G_z$ to $+8G_z$ on 35 subjects, except immediately before blackout. These results are difficult to reconcile, in view of later work by Brown and Burke [25] (1957) and by Canfield et al.[33] (1949), in all of which delayed reaction time was observed. Frankenhaeuser [69] (1958) carried out a very definitive test in which she exposed subjects to $+3G_z$ while timing their response to a five-choice situation. The subjects were experienced in acceleration and trained in the task. Reaction times during exposure to +3G were significantly longer than under normal conditions; however, the effect was more pronounced during the first 2 minutes of a 4-minute period than during the last 2. As J. L. Brown [24] (1961) points out, there is a wide variability in the effect of acceleration on reaction time, and it seems reasonable to

believe there is an actual increase. The discrepancies in results may well be related to different interpretations on the part of different investigators.

The situation is again complicated by the work of Canfield et al.[32] (1950), who investigated the time taken by subjects to determine the direction of moving lights and found that after an initial learning period there was no difference in time under accelerations of 3 and 5G.

Work by Chambers and Hitchcock[36] (1963) showed that the response time for discrimination of colored lights was longer under G_z acceleration than under normal conditions. During a 5-minute exposure to $6G_x$, however, the response of each subject to 25 trials of light discrimination was slower than average; during a second 5-minute exposure it was still slower, but during the third series performance improved significantly. This suggests that the subjects had learned to adapt to the acceleration stress. A similar variation in response time was observed with auditory stimuli.

Because of the importance of perceiving and interpreting signals and instruments, some definitive resolution would be of value.

To recapitulate the foregoing, it would appear that sustained acceleration reduces the visual field; diminishes voluntary eye movements to the point of ataxia; impairs the detection of low-intensity signals, differences in brightness, and the resolution of fine detail; and interferes with reading tasks in moderate to low luminance. Accommodation is apparently unaffected and the question of visual reaction time is not completely resolved.

VESTIBULAR AND KINESTHETIC SENSES

Examination of the role of the vestibular and kinesthetic senses in acceleration leads into a consideration of the whole field of motion sickness, illusions, the effects of a rotating environment, and spatial disorientation.

AUDITORY SENSE

It is well established that the sense of hearing is maintained after acceleration has reached a magnitude sufficient to cause blackout, although it does not appear to have been experimentally established whether loss of hearing occurs prior to, or with, unconsciousness. The point is not entirely academic; it is of practical importance to know whether an auditory warning will be heard after a visual warning is no longer perceived. Such evidence as there is indicates a progressive increase in reaction time to auditory stimuli as unconsciousness approaches (Canfield et al.[33] 1949). Again, however, this increase in reaction time to auditory stimuli is denied by Franks et al.[71] (1945).

BODY MOVEMENTS

Gross body movement is progressively impaired with increasing acceleration. Code et al.[50] (1947) found that walking, crawling, and movement along a ladder against acceleration were very difficult at $+2G_z$ and impossible at $+3G_z$. Movement at right angles became impossible at $+4G_z$; parachute donning time was increased from 17 seconds at 1G to 75 seconds at $+3G_z$. At $+6G_z$ to $+7G_z$ it is extremely difficult to reach a face-curtain seat-firing handle (Christy,[39] 1961).

In the $+G_x$ position, the body, legs, and arms cannot be lifted at $8G_x$ and above. The unsupported head cannot be lifted above $9G_x$, although use of a counterweighted headgear allows relatively free movement up to $12G_x$ (J. L. Brown,[24] 1961). Hand and wrist movement seem to be possible up to about $25G_x$. The free movement permitted in water has already been noted.

CONTROLS

Little is known about muscle response under acceleration. However, using an ingenious electromyographic technique, but only one subject, Wells and Morehouse[180] (1950) analyzed the response of arm muscles during 10 to 50 lb pulls on an aircraft control stick under $+1G_z$ to $+5G_z$ with the arm in flexed, intermediate, and extended positions. They found that voluntary muscular exertion increases with increase in acceleration in a manner that is just sufficient to balance the change imposed by acceleration. This would

appear to be the only study of its kind, and more information would be of value on the limits of force that can be exerted with different muscles under different acceleration conditions.

Because of the forces engendered by acceleration, control maneuvers involving gross motions of limbs are not possible, and control must be mediated by wrist and arm action. Brissenden [21] (1957) examined the forces which pilots could exert on a side control stick in the 1G situation, but did not study the modifying effects of added acceleration. Movement of hand controls is impaired relatively little, however, at high accelerations. Collins et al.[52] (1958) showed that pilots could operate a right-hand control stick and a thumb switch on a contour couch up to $+25G_x$.

Chambers [34] (1961) lists the characteristics that should be borne in mind in designing control sticks

1. Stick force gradients along each mode of control
2. Centering characteristics along each mode of control
3. Break-out force
4. Control friction
5. Damping characteristics
6. Control throw
7. Control response time
8. Control harmony
9. Cross coupling
10. Control feedback
11. Control sensitivity

There is as yet no ideal stick control, as is evident from the observations of Chambers, who has done much of the work in this field, although many of his papers reflect the same data. In a paper discussing the problems of side arm controllers, along with a large amount of generalization, Chambers [34] (1961) makes reference to the testing of seven different types of three-axis or two-axis side arm controllers, and illustrates the performance error found with some of them. Unfortunately, perhaps because much of the source material is classified, it is not possible to get a clear grasp of the advantages of one type over another. There is no doubt, however, that there is a wide variation in the efficiency of different types of side arm controls, under different combinations of acceleration vectors. An example is shown in figure 39, which illustrates pilot control proficiency under various combined acceleration fields with different types of hand controllers. Note that the abscissa refers to combinations of applied fields, and not acceleration resultants. In this situation longitudinal and lateral period of motion and damping were kept constant, and the score gave emphasis to the longitudinal control task. The pilot proficiency score, however, does not neces-

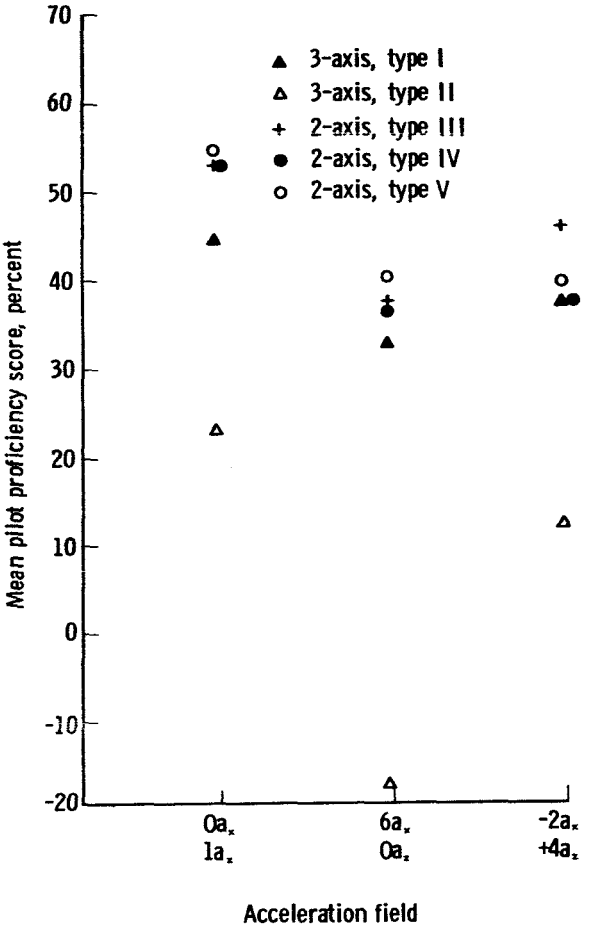


FIGURE 39.—*Mean pilot efficiency scores in different acceleration fields.* (Chambers,[34] 1961.)

sarily match the pilot rating for similar circumstances, as shown in figure 40. Even wider differences in stick performance are shown in the response to roll (fig. 41). It would appear that much could yet be done in the selection and testing of manual control systems.

Little work has been done on the use of foot controls under high acceleration. Clark et al.[44] (1959) point out that at $+5G_x$ it is difficult to hold the feet forward on rudder pedals. However, in the contour-couch position, use of dorsiventral rotation at the ankles has some promise, as was shown by Smedal et al.[156] (1960).

CEREBRAL FUNCTION

Loss of consciousness and complete impairment of cerebral function occurs between $+3G$ and $+8G$ in the G_z vector, the specific level depending chiefly on biological factors, duration, and rate of onset. In the other vectors the subject normally reaches a tolerance threshold of another sort before unconsciousness occurs. On return to consciousness there is usually a short (5 to 15 second) period of confusion.

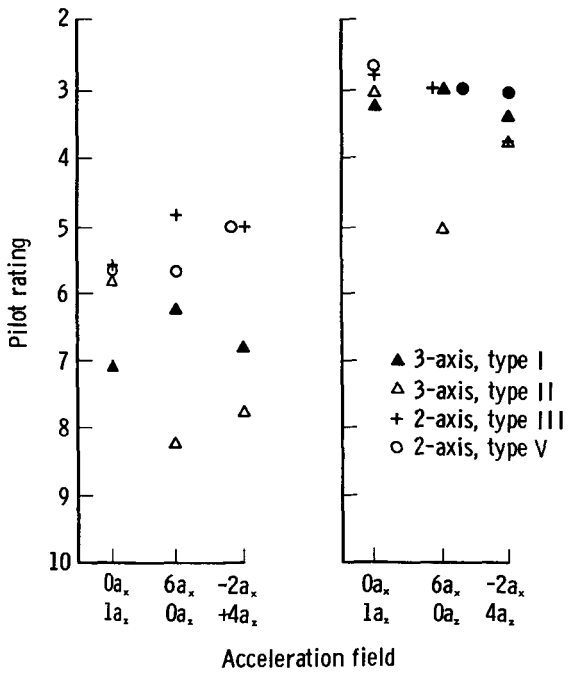


FIGURE 40.—*Mean pilot ratings of vehicle controllability with several side-arm controllers.* (Chambers,[34] 1961.)

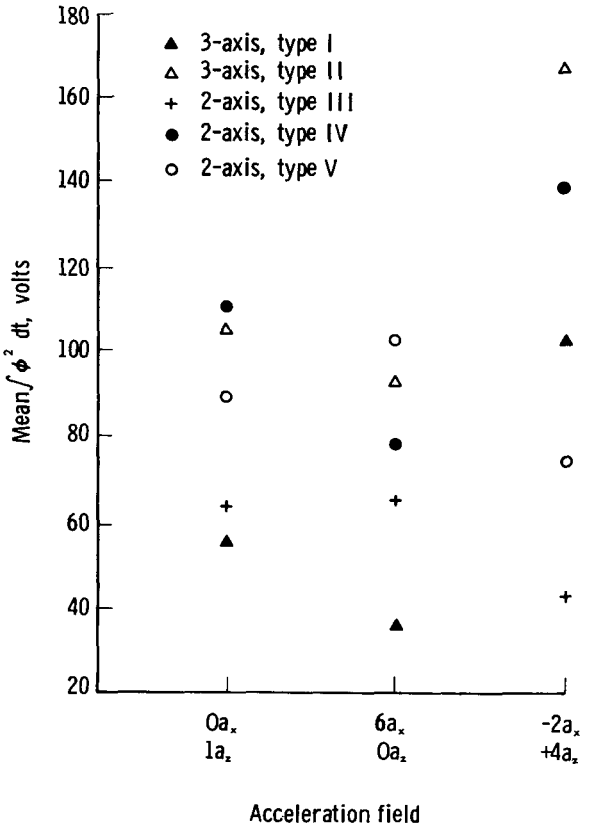


FIGURE 41.—*Roll error scores for pilots who performed a tracking task within several acceleration fields.* (Chambers,[34] 1961.)

It is of importance to know, however, whether there is impairment of cerebral function before the onset of unconsciousness. In response to any form of functional testing some factor of cerebral function is involved. Thus, much of the testing previously noted includes an examination of certain levels of cerebral function.

The estimation of flicker fusion frequency is one such measure, and although involving the eye, it also provides an estimate of the integrity of neural paths. Keighly et al.[103] (1951) examined flicker fusion frequency with and without extraocular negative pressure. In the former case there was no change up to the point of grayout ($+3.2G_z$). In the

latter, at $+4.8G_z$ the frequency dropped from 43.71 to 42.36 cps, a difference significant at the 0.01 level. From this the authors concluded that there was impairment in the cerebral circulation, in addition to impairment of the retinal circulation, since the latter could be improved by application of negative pressure.

There is relatively little data, however, on higher mental functioning. Subtraction and multiplication tests were presented to subjects at $+3G_z$ for 2 to 10 minutes by Frankenhaeuser [69] (1958), who showed that time taken to complete a task of this type increased with the acceleration applied. Chambers [35] (1963) quotes a study by Cope et al., who used an auditory mathematical presentation and a press-button response for their test, and again found a delayed response with acceleration.

On the other hand, Wilson et al.[187] (1951) exposed subjects to seven 1-minute trials at $3G_z$, evaluating color-naming ability, arithmetic, steadiness, tapping, number ranking, and word separation. No significant effect was observed except in color-naming and steadiness, where performance was poorer under acceleration. Perhaps the difference lies in the shorter duration of exposure.

In testing other complex skills, Frankenhaeuser [69] (1958) demonstrated a significant decrement in color-naming under acceleration. Chambers [35] (1963), using a complex test involving continuous and repetitive memorization of a portion of a sequence of random numbers, found that his subjects could perform this task as well at $+5G_x$ as at 1G, but the subjects stated that the mental strain was much greater at $+5G_x$. In another task, monitoring a changing display of numbers and symbols and matching the current display with one presented some time before, Chambers found that immediate memory was unaffected to $+5G_x$ but impaired at $+7G_x$ and above.

On the basis of these various tests it would appear that higher function is disrupted under acceleration, at least to the extent of interference with concentration. Most of the color-naming, word separation, and mental arithmetic types of tests are of dubious value. It would be useful, however, to investigate the higher mental function during different magnitudes, vectors, and particularly durations, of acceleration, by means of a series of perceptual or cognitive tests after the style of Chambers.

TRACKING TASKS

Probably the most effective meaningful measure of complex function lies in tracking and instrument monitoring. Creer et al.[54] (1962) investigated the ability of trained subjects to perform a complex tracking task on the Johnsville centrifuge under varying conditions of G-load, vector, duration, and rate of onset. They observed a marked deterioration in tracking ability immediately after onset of acceleration, which was attributable to the angular rotation of the gondola. This emphasizes a point raised by J. L. Brown [24] (1961), that while a centrifuge can simulate an acceleration field, the motions through which it goes to achieve this are unlike those of an aircraft or spacecraft and may modify a performance response. However, after the deterioration, noted above, tracking performance improved, and stabilized at a constant level. The results showed that within the parameters simulated in this study a pilot could adequately control his vehicle in a field of $+14G_z$. Best control was in the $+G_x$ vector, next in the $-G_x$, and least in the $+G_z$. With rates of onset varying from 0.1 to 2G per second, tracking performance declined above 0.75G per second. Even with the Ames restraint system, Smedal et al.[155] (1963) found tracking performance during $-G_x$ conditions was no better than during $+G_z$ despite the easier respiration.

In connection with the Military Astronaut Training Program, Chambers and Nelson [37] (1963) described the results of a simulated task representing control of an undamped vehicle by three-axis proportional control during a rise time of 15 seconds to various peak accelerations, each of which was maintained for 125 seconds. The results, sum-

marized in figure 42, clearly show the advantage of the $+G_x$ vector in achieving best performance.

These experiments of course were undertaken in conditions of sustained acceleration. Other tasks involve exposure to changing acceleration such as would be found in exit and reentry of a space vehicle. An example of this type of study is that performed by Preston-Thomas et al.[138] (1955), who exposed nine subjects to a three-stage profile

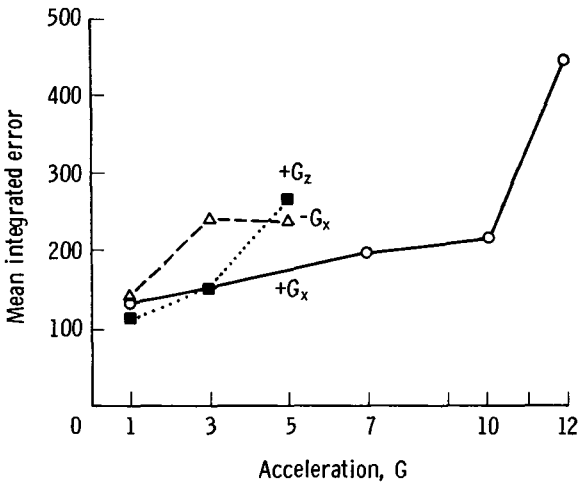


FIGURE 42.—*Comparison of the effects of direction and amplitude of acceleration upon pilot error of 12 test pilots in a three-axis rate-damping task.* (Chambers and Nelson,[37] 1963.)

(fig. 35) and determined their efficiency in controlling a two-dimensional compensatory tracking task. The results showed some degradation in performance, but from the data it is not possible to quantify the degradation with any refinement. Decrement was again noted in association with angular acceleration.

Smedal et al.[154] (1960) described a further tracking task of this type where pitch, roll, and yaw inputs were presented on an oscilloscope. The subject responded by the use of a pencil-type controller for pitch and roll, and toe pedals for yaw. Unfortunately the performance data are not presented in the paper, although the authors state that "a well trained subject . . . can successfully perform a moderately complex tracking task, representative of a reentry control problem, while being subjected to relatively high and varied accelerations for prolonged periods of time."

The greatest refinement in the type of task comes when pilot control inputs are permitted to modify the centrifuge dynamics to simulate actual control of a vehicle. Chambers and Holloman (Chambers and Hitchcock,[36] 1963) exposed pilots to a staging type of acceleration characteristic of a two-stage and a four-stage launch vehicle, arranging the controls so that pitch required almost continuous control whereas yaw required monitoring and occasional correction. They found little impairment up to the $7G_x$ limit of their studies, but the subjects observed that under acceleration they could concentrate on only a portion of the task requirements at a time.

One unusual form of environmental performance testing involves high-speed rotation. Weiss et al.[179] (1954), in their study of simple tumbling, undertook some gross performance testing by presenting visual and auditory signals which required a press-button response. No errors were observed up to 100 rpm with center of rotation at heart level, and no increase in reaction time was noted.

In another test of motor performance the subject was required to simulate a manual ejection sequence. A very slight increase in reaction time was noted. In a rotary field of changing rate, however, the subjects had difficulty in locating a toggle switch so placed that it could not be seen. Using the NASA Multi-Axis Test Facility, Useller and Algranti [171] (1963) exposed trained pilots to complex rotations, producing a resultant of up to 70 rpm, and tested their ability to counteract the induced rotation by activating jet nozzles. Up to the limits studied, the pilots were able to perform their task with an error ranging from 6.5% to 18%, depending on the training and skill of the individual. Within the range measured, the rate of rotation did not affect performance. In this test, however, error was evaluated as the percent-

age of the total time the subject made an incorrect input, but the error score did not account for errors of omission. It would be of value to determine the performance on a timed basis, with higher resultants, and to include an imposed acceleration field.

Simulation of specific space missions also provides opportunity for performance study. Within the capacity of the simulator and the accuracy of the theoretical parameters of the mission, this can give a fair indication of the subject's ability to perform a specific mission, while at the same time providing a degree of training.

In one portion of their somewhat general paper, Chambers and Hitchcock [36] (1963) describe the results of 12 simulated Mercury missions, 8 of which included centrifuge motion combined with various other Mercury parameters. Three general effects were noted in the course of this program:

1. Acceleration resulted in the insertion into the system of inadvertent control inputs. These inputs normally occurred in one axis, as noted in figure 43, but occasionally occurred in more than one. Such inadvertent inputs may be associated with excessive fuel utilization.

2. Acceleration generally disrupted the timing and precision of pilot control, although it would appear that the disruption which occurred, while obvious on the recording instruments, was not great enough to have a critical effect on the final adequacy of the performance.

3. Discrete task functions, such as an operation override, were affected by accelerations which preceded and/or followed them, though the operations themselves were performed under minimal acceleration loads.

Chambers and Hitchcock also report that during dynamic simulation (that is, in closed-loop operation of the centrifuge where the pilot input is reflected in centrifuge reaction), the overall mean response time to tally panel indications represented by a "red light" or a "no light" indication is more variable than in open-loop operation. Furthermore, the response time was considerably longer when a "no light" indication was given. On very few occasions, however, did the pilots fail to make any response.

Alexander et al.[3] (1964), in his study of oxygen saturations during Apollo type profiles, also gave his subjects a performance task. Using a three-axis wrist controller and an "eight-ball" type of display, they performed a task simulating control of a vehicle during the acceleration phases of its mission. Error in three planes was integrated over time. Significant decrement in performance occurred during the simulated missions but it was not possible to say how much of the decrement was associated with the desaturation and how much was due to the acceleration per se. The next approach, of course, should be repetition of the task in a 1G environment with atmospheres artificially varied to produce the same degree of desaturation found under G_x acceleration.

In the end, the proof of the pudding is in the eating, and successful completion of their missions by the astronauts, frequently under exacting circumstances, attests to the fact that man can perform adequately under the conditions of sustained acceleration found in Mercury launch and reentry and indicates the likelihood of equally adequate performance in still more stressful situations.

This, however, should under no circumstances be interpreted as implying that serious consideration of further protection

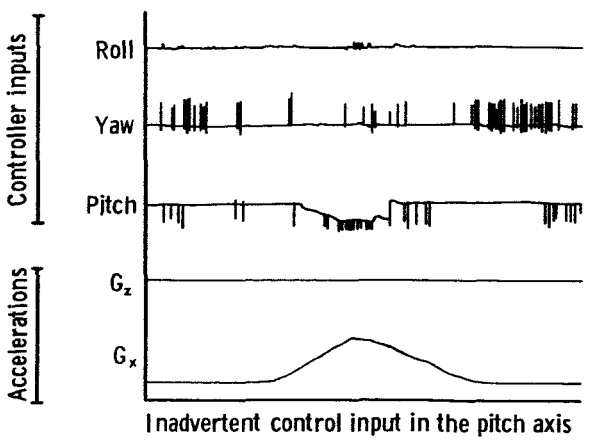


FIGURE 43.—*Inadvertent control input in the pitch axis.* (Chambers and Hitchcock,[36] 1963.)

against the effects of sustained acceleration is unnecessary. In emergency situations, and with high motivation, man can tolerate extremes of stress for a short period with apparently minimum decrement. Wherever possible, however, protection, particularly against the expected degree of arterial hypoxemia and the associated possibility of structural damage to the lung parenchyma, is highly desirable if not mandatory.

CHAPTER 6

Conclusions and Developments

From the preceding review, certain broad conclusions can be drawn, or perhaps reaffirmed, since most of them are not new. The basic physiological response to sustained acceleration in all vectors depends primarily on the development of a hydrostatic pressure head related to the magnitude of the acceleration. Despite protection by body compensatory measures, this pressure head interferes grossly with blood circulation. The development of compensatory measures requires a significant time, of the order of 5 seconds, and consequently is influenced by the rate of onset of acceleration. The nature of the manifest interference depends on the acceleration vector. In the positive and negative ($\pm G_z$) vector, the major effect is observed in the retinal and cerebral circulation; in the transverse ($\pm G_x$) vector, the subjective effects are chiefly respiratory, although the development of alveolar shunts is probably physiologically more significant; while in the lateral ($\pm G_y$) vector, again it is probable that physiological shunts and arterial desaturation may be significant.

The cellular response is probably not specific and appears to represent the response to hypoxemia or to general stress.

From the pragmatic point of view, human tolerance and the ability of man to perform a given task under sustained acceleration are of primary interest. Each of these is dependent on the interplay of a multitude of factors, many of which do not lend themselves to measurement, and consequently only general approximations can be defined. However, numerical definitions of some of these capacities have been described, and become the more useful when interpreted in the light of how they were obtained. One cannot, for example, blindly assume that since man can withstand an exposure of $17G_x$ for 1 minute, a vehicle can be designed to exploit this capacity. It is true that under certain circumstances a highly motivated man can tolerate $17G_x$ for 1 minute and even perform certain maneuvering actions, but the circumstances and the man must be exceptional.

Equally, we cannot adequately define performance capacity under sustained acceleration until we define the performance requirement and are able to measure performance, particularly higher function, in a satisfactory manner under nonstressful conditions.

The development of methods and devices for protection and restraint goes hand in hand with raising or maintaining the threshold of tolerance and performance. It would appear that we have virtually surpassed the usefulness of the pneumatic anti-G suit, although the possibilities of posture, water immersion, and pharmacological agents have not yet been fully exploited.

Thus, in spite of the vast amount of work that has been done in this field, there are still areas where little or no data are available, and others where the data are inadequate for a full definition of the response or the limiting threshold. Many of these have been mentioned in the course of this text, and some will be reiterated and examined in this section.*

UNCOMMON VECTORS

It is probably not surprising, in view of the practical applications, that the effects of sustained acceleration in the lateral ($\pm G_y$) vector and diagonal vector have been largely unexplored. Although there are no urgent requirements, it is believed that these areas should receive a definitive examination, if only for completeness. It is possible that with

* This study was completed early in 1964. Since then, work has been started in several areas about which recommendations are herein made.

suitable restraint, the $-G_x$ or the $\pm G_y$ vectors will be found more tolerable, and that the respiratory and saturation problems may be less severe than those in the $+G_x$ vector. In the negative ($-G_z$) vector, also, there is a need for a general reexamination and extension of the work which was originally initiated but not pursued, particularly in the cardiorespiratory field.

VARIABLE-RADIUS CENTRIFUGE

A variable-arm centrifuge with a radius variable from zero outward has much to commend it. Such a device would combine the advantages of a rotating chamber, a short-arm centrifuge and a long-arm centrifuge. The practicality and usefulness of a short-arm centrifuge requires investigation, along with an evaluation of the minimum length of arm, in view of engineering limitations, that might conceivably be employed in a space vehicle. From an academic point of view it would be of value to study the pressure gradient that would develop along a body axis when a subject is accelerated on a short-arm centrifuge. Equally it would be useful to distinguish as much as possible between the effects of radial acceleration and combined radial and tangential accelerations. All of these could be investigated on a variable-arm centrifuge.

VERY PROLONGED ACCELERATION

With the exception of the 24-hour study of Clark [40] (1960), nothing has been reported on the effects on man of exposure to longer than a few hours' acceleration, and even Clark's study was more in the nature of an initial observation. It is believed that exposure to very prolonged relatively low acceleration may become an increasing requirement, and definitive investigations should be initiated, to include studies of the physiological effects of, tolerance to, and performance in, low-acceleration fields. Magnitudes should range to perhaps 2G or higher, for periods of several months where possible.

RATE OF ONSET

Another relatively unexplored field which has tacitly been recognized as significant, although little in the way of definitive attack has been made on it, is the effect of varying rate of onset. In impact studies the effect of rate of onset is well recognized, but much less attention has been paid to it in considerations of sustained acceleration. There is probably an optimum rate for different peaks and plateaus that allows development of maximum compensatory measures with the minimum increase in the impulse. Alternatively stated, there are probably optimum combinations of rate of onset, offset, peak, and plateau to provide the most tolerable change in velocity compatible with engineering limitations. These combinations have not yet been determined. Engineering considerations are vital here, and for some time to come they may well dictate many of the parameters. However, within the limiting envelope there is much that can yet be done.

CARDIORESPIRATORY STUDIES

There is no doubt that cardiorespiratory problems are the chief limiting factors during exposure to sustained acceleration. The particular manifestation, of course, depends on the vector, the magnitude, and the duration.

In the $+G_x$ vector, dyspnea, pain, and cough are the chief subjectively limiting factors, although the accompanying arterial desaturation is probably of more physiological significance. Obviously much has yet to be done in establishing the nature and degree of the associated shunt. In this regard, regional studies in all vectors would be useful, and techniques employing radioactive xenon for ventilation studies and tagged albumen for perfusion studies would be of particular value. Diffusion studies are also needed. The slower recovery of arterial oxygen saturation in men and dogs breathing oxygen under $+G_x$ acceleration as opposed to breathing air has a bearing on the choice of cabin atmospheres, and needs further investigation.

Gas exchange under acceleration has not been definitively elucidated. The work of Zechman et al.[197] (1960) has shown an increase in oxygen consumption with $+G_x$ acceleration, and this has been demonstrated

by others, but with the techniques used, and probably unsteady conditions, the picture is not clearly defined for any vector.

The investigations into breathing mechanics under both positive and transverse acceleration, although more definitive, are still incomplete. The occurrence of persistent hyperpnea needs further examination with respect both to its cause and to its relationship to hypercapnia and possible disturbance of pH. Consideration of altered lung volumes and pressure-volume relationships raises the question of developing and using techniques for determination of regional compliance. This would aid in determining the extent of physical pulmonary changes.

The effect and usefulness of pressure breathing require still more examination. The work of Watson and Cherniack [174] (1962) has shown its subjective value under certain environmental conditions, but more work is needed to determine the value of pressurized air as opposed to oxygen, the effect of varying ambient pressures, particularly those used or contemplated in space cabins, and the effect of varying atmospheric combinations. The optimum positive pressure and the time before onset of acceleration when this positive pressure should be applied for maximum effect also need to be defined.

Cardiological problems, particularly in the $+G_z$ and $+G_x$ vectors, have received much attention, notably from the Mayo group, and definitive results have been obtained—although the accuracy of the techniques has been questioned. Not yet established, however, is whether the cardiac compensatory measures are maintained indefinitely, or whether after a certain time at a given acceleration there is a breakdown of compensation and eventual loss of arterial pressure and unconsciousness. This area requires investigation.

In this regard, too, the convulsions that may occur with unconsciousness under G_z acceleration need more investigation. Why do they occur in some and not in others? Are they similar to the convulsions of grand mal epilepsy? Do they imply an epileptic diathesis or a persistently low threshold for epilepsy? Why are they not accompanied by incontinence?

In other vectors, cardiological problems have received less attention. The question of arterial rupture during $-G_z$ acceleration remains unresolved. Is it inevitable if the magnitude is great enough, or does it depend upon a congenital weakness at a point in the cerebral arterial tree?

In the $-G_x$ and $\pm G_y$ vectors no cardiac studies, other than ECG, appear to have been done. For completeness, these should be undertaken.

The question of ECG's under acceleration opens a broad field of cardiac examination using some of the newer techniques of vibrocardiography which show much promise, although the difficulties of obtaining records of this type under acceleration are very great.

CELLULAR PHYSIOLOGY

Little emphasis has been placed on the cellular physiology of sustained acceleration. However, it would appear likely that response on a cellular level is closely related to the response to stress, and in fact may be simply a manifestation of a general response to stress. The work of Polis et al.[136] (1962) and Stiehm [167] (1962) in this regard, while not completely definitive, and open to interpretation, represents an approach to this problem and deserves support.

RENAL PHYSIOLOGY

The effect of sustained acceleration on renal output is of course only one particular consideration within the whole field of renal function and blood volume control. Acceleration, however, is a useful tool for varying effective blood volume in the atrial region, and as such could be effectively used in ADH studies. In particular, studies at $-G_z$ would appear to be very useful. Information on the specific effects of acceleration is somewhat equivocal, or a least inadequate, and further definitive studies in all vectors are needed. With identification of the effects of acceleration, more information will become available on the nature of the volume-sensing mechanism and the hormonal control.

TOLERANCE STUDIES

Although data points are available to provide approximate definitions of maximally tolerable conditions, there is little definitive information to serve as a basis for delineating reasonable working limits for the ordinary healthy man. Empirically, it would appear that an ordinary individual can withstand and perform satisfactorily during exposure to $+2G_z$ to $+2\frac{1}{2}G_z$ or $+3G_x$ to $+4G_x$ for periods up to half an hour with suitable restraint. For periods up to about 1 minute, the maximum working level for ordinary purposes appears to be about $+10G_x$. However, these numbers are somewhat arbitrary. It would be of considerable value for design purposes to validate these figures over a broad population.

It should also be noted that no tolerance data, or, for that matter, general acceleration data, are available for females.

Discussion of tolerance requires a consideration of objective end-points. As has been noted, there is a requirement to develop and standardize objective end-points. This is a difficult problem, and although several solutions have been suggested none as yet is entirely satisfactory. It is probable that the best solution in the $+G_z$ vector may be found in the EEG, whereas in the $+G_x$ vector desaturation levels may prove useful.

As will be noted from figure 26, there are still areas where data points are very sparse. In particular, in the $\pm G_x$ vector information is needed on the maximum tolerance for durations up to 5 seconds, while in the $+G_z$ vector data are required for tolerance up to $1\frac{1}{2}$ seconds. A "whiplash" type of device could be designed to investigate this area. At the opposite end of the scale, tolerance data are required in all vectors for durations beyond 20 minutes. Data for all durations are required in the $\pm G_y$ and $-G_z$ vectors.

The question of fatigue and the relationship between exercise fatigue and acceleration fatigue deserve a systematic study, which should be enlarged to include examination and comparison of the fatigue associated with other forms of environmental stress such as hypoxia, vibration, and buffeting, noise, thermal exposure, and so on. It would appear that, while related, the fatigue associated with different forms of environmental stress is not the same as that associated with exercise, even under circumstances in which the exercise involved might be adjudged as being equivalent in each case.

Little more has been accomplished in evaluating tolerance in a rotational field, and under combined accelerations, since Weiss et al.[178,179] carried out their studies in 1954. In view of the possibility of catastrophic rotation in a space vehicle, it is believed that this whole field should be reopened. In particular, studies such as those of Useller and Algranti[171] (1963) should be pursued.

Tolerance thresholds are of course influenced by the nature of the restraint and protection available. As has been pointed out, there is need for development of a multifitting restraint system suitable for all vectors. The custom-molded contour couch and the Ames restraint system are awkward and cumbersome, and for that matter, the optimum angles of head, torso, thighs, and legs have not yet been fully established or implemented. It is believed that much can yet be done with slings and nylon net. The airbag system of Clark[41] (1964) is not very compatible with the other engineering requirements of a space cabin, but it has promise and deserves continued investigation.

The use of water immersion has been allowed to drop since the initial dramatic success of Gray and Webb[88] (1960). It is obviously not practicable at this time to utilize a water tank in a space vehicle, but much more work is required to define physiological effects, tolerance, and performance in a water immersion system and it might be very advantageous to determine anew the usefulness and practicality of a fluid-filled suit with the fluid as an integral part of the environmental control system.

Complete submersion and water breathing remains in the realm of laboratory animal experiment. The work of Black-Schaffer et al.[17] (1961), however, has considerable academic interest and should be pursued.

Support also is due the work of Polis[135]

(1961) on pharmacological protection. Apparently the only study of its type, this has a great deal of promise. There is little doubt that other agents will be found which will not only increase tolerance, but may provide a key to some of the cellular mechanisms involved in the response to acceleration.

PERFORMANCE STUDIES

There are two major approaches to performance testing under environmental stress. One is to simulate as closely as possible the actual required task and measure the ability of the subject to implement it. This has been done with some degree of success. This somewhat pragmatic approach, however, does little to elucidate the effect of the stress on specific sensory, motor, or cerebral modalities. The other approach is to attempt to isolate and measure the responses of the specific modalities in a quantitative and qualitative manner. Unfortunately, the results may not always be very meaningful, since all too often it is not clear precisely what one is measuring.

Even with this limitation, however, much can yet be done. Little information is available on color vision or on the effectiveness of peripheral vision prior to grayout. The apparent continuum of progressive visual failure has not been fully established. Methods of improving vision in the $-G_x$ vector need development and evaluation. Eye movement and pupillary reactions need further study, and the effect of acceleration on visual reaction time is not fully resolved. In addition, methods might be investigated for the development of G-sensitive rheostats for the control of lighting under acceleration.

With regard to hearing, only gross examinations have been made under acceleration. Audiometry might well show as yet unconsidered changes, while the actual relationship of loss of hearing to loss of consciousness remains to be established.

With the exception of the vestibular apparatus, the other sensory modalities remain virtually unexamined.

A definitive study of muscular coordination and the ability to utilize different muscle groups within a broad spectrum of acceleration magnitudes, vectors, and durations would be of considerable value in a reconsideration of controller devices, particularly since the designs of these devices were at least partly based on a relatively small number of user opinions.

A further development in control systems that bears watching is the current investigation, by the Philco Corporation and New York University, of the feasibility of utilizing electrical potential on the surface of the skin for activating controls. This could have considerable value, when developed, in maintaining control of a vehicle during exposure to high-intensity acceleration.

The most significant area for consideration, however, is probably that of higher mental functioning. Measures of intellectual ability, concentration, judgment, decision-making, and the like are notoriously difficult to implement. Computations and word-games are of doubtful value as testing procedures because they depend so much on preestablished patterns and abilities. Chambers [35] (1963), however, has made a valuable approach to this problem which should be followed up.

Annotated Bibliography

As noted in the preface, this annotated bibliography has been prepared instead of a reference list.

While it embraces all references cited in the text, it also includes certain other abstracts which are considered appropriate or which, for one reason or another, were not incorporated into the textual material. Since it is, in fact, an expanded reference list, it should not be considered as a comprehensive bibliography. For such the reader is referred to Research Bibliography No. 43, Physiological and Psychological Effects of Space Flight, Volume 1, Space Technology Laboratories, Inc., October 1962.

Most of the following abstracts were prepared by the writer in connection with developing the text; some, however, are authors' summaries, and some are extracted from the above noted bibliography.

1. AGADZHANYAN, N. A., MANSUROV, A. R.
   The effect of oxygen deficiency and prolonged radial acceleration on an animal organism.
   Bull. Exp. Biol. and Med. (Moscow), *53*:42, 1962. (Translation)

Authors measured the response of dogs to altitude (approximately 2,000 to approximately 30,000 ft) and to acceleration ($+2G_x$ to $+9G_x$) using a short-radius (approximately 12 ft) centrifuge. With respect to acceleration, X-ray showed reduction in heart size, decrease in shadow intensity, and changes in lung appearance. Substantial changes were observed in thoracic organs by X-ray, even at 2 to $4G_x$ in anesthetized animals, while similar changes were observed at $6G_x$ or more in nonanesthetized animals.

2. AGOSTINI, E., THIMM, F. F., FENN, W. O.
   Comparative features of mechanics of breathing.
   J. Appl. Physiol., *14*:679, 1959. (Authors' Summary)

Dogs, cats, rabbits, guinea pigs, and rats have been studied. The lung-thorax compliance per unit body weight and per unit vital capacity shows some significant differences between species. Static pressure required by a normal inspiration is, however, similar in different animals. The expiratory reserve per unit vital capacity and the functional residual capacity per unit total lung capacity are larger in the animals breathing at lower frequency. These findings are related to the hypothesis that animals breathing at lower frequency have a larger functional residual capacity in order to contain within small limits the changes in composition of the alveolar air due to the breathing cycle. Relationship between dynamic pressure and flow follows Rohrer's equation. The dynamic pressure required by a normal ventilation is similar in the different species. Relationship between rate of work of breathing and breathing frequency shows that the frequency typical of each animal is that corresponding to the minimum rate of work.

3. ALEXANDER, W. C., SEVER, R. J., FEDDERSON, W. E., HOPPIN, F. G.
   Acceleration ($+G_x$) induced hypoxemia and crew performance.
   Aerospace Med., *35*:257, 1964. (Authors' Summary)

A study of arterial oxygen saturation and pilot performance during sustained accelerations similar to those anticipated in the Apollo and subsequent earth entry missions has been conducted in a joint Manned Spacecraft Center-Aviation Medical Acceleration Laboratory program at the Johnsonville centrifuge facility. Cabin environments of one atmosphere and one-third atmosphere, with air and 100 percent oxygen, respectively, breathed on demand by the pilot were employed in the study. Data collected from 25 professional military pilots demonstrates a resultant diminishing arterial oxygen saturation as a function of magnitude and duration of acceleration and the environment of the pilot. A deterioration in pilot performance was also demonstrated, the extent being dependent upon the severity of the programed accelerations.

4. ANDINA, F.
"Schwarzsehen" als Ausdruck von Blutdruckschwankungen bei Sturzflugen.
Schweiz. Med. Wchnschr., 67:753–756, 1937.

5. ARMSTRONG, H. G.
Principles and Practice of Aviation Medicine.
Williams and Wilkins Co., Baltimore, 1939.

6. ARMSTRONG, H. G., HEIM, J. W.
Effect of acceleration on living organisms.
J. Aviat. Med., 9:199, 1938. (Authors' Summary)

Positive accelerations, directed through the long axis of the body and acting in the direction seat to head, result in a shifting of the blood away from the head region to the lower portions of the body. This shifting of the blood away from the head produces a cerebral anemia in the average normal young male adult such that at forces equal to about 5 times the pull of gravity there occurs a temporary loss of vision and at forces of 5 to 7 times the pull of gravity there occurs a loss of consciousness.

There is a definite timelag between the onset of positive accelerations and a shifting of the blood mass such that about 5 seconds are required from the time any given acceleration is reached until the full physiologic effect is obtained. As a consequence the shorter the duration of any acceleration (under 5 seconds) the less the physiologic effect.

There is a wide variation in individual susceptibility to positive accelerations which is probably due to the variations in vascular tone and vasomotor control in different individuals. Tolerance is decreased by rapidly repeated exposures but increased by repeated daily exposures. Tolerance may be moderately increased by tensing the muscles of the legs and abdomen and markedly increased by applying 100 mm Hg pressure to the abdomen by means of an inflatable belt at least 30 seconds prior to the acceleration.

Accelerations of +12.2G in flight and +16G in the laboratory have been tolerated without evidence of permanent injury.

Negative accelerations, directed through the long axis of the body from head to seat, cause a shifting of the blood away from the lower portion of the body to the head region. This shifting of the blood to the head region results in a high intracranial blood pressure such that at about −3G symptoms of concussion and at about −5G massive cerebral hemorrhage and death may result.

There is little timelag in negative acceleration, there is no tolerance developed, and there is no obvious method of counteracting its effects.

Transverse accelerations, directed through the body perpendicular to its long axis and in any direction, are relatively well tolerated up to 12 times the force of gravity. The principal physiologic effect of high transverse accelerations of 8G and above is a compression of the supporting framework of the body trunk, tending to induce forced expiration, which is normally counteracted by an involuntary holding of the breath in mid-inspiration.

7. BALLINGER, E. R.
Human experiments in subgravity and prolonged acceleration.
J. Aviat. Med., 23:319–321, 1952.

The physiological problems of weightlessness and acceleration are presented. Experiments were run on the human centrifuge at the Aero Medical Laboratory to determine the optimum acceleration one could safely undergo in attaining an escape velocity.

The author exposed nine subjects, in the supine or semisupine position, to sustained acceleration with chief complaints as shown in the following table:

G	Time	Runs	Failed	Complaints
		Supine position		
3	9m 31s	9	1	Monotony during acceleration, giddiness after
4	6m 21s	9	0	Giddiness after acceleration
5	4m 45s	9	0	Substernal discomfort
6	3m 48s	7	1	Severe substernal discomfort
7	3m 10s	8	1	Substernal pain and dyspnea
8	2m 40s	17	7	Severe substernal pain and dyspnea
		Dorso-sacral position		
8	2m 40s	5	0	Epigastric discomfort
9	2m 20s	3	0	Severe epigastric discomfort and dyspnea
10	2m 6s	3	1	Epigastric pain and severe dyspnea

8. BARER, A. S., GOLOV, G. A., SOROKINA, YE. I.
Physiological reactions of the human body under the influence of acceleration of critical duration and magnitude directed along the back-chest axis.
Bull. Exp. Biol. and Med. (Moscow), vol. LVI, no. 8, Aug. 1963, pp. 33–37.

Authors studied some aspects of respiration during accelerations up to $+15G_x$. They demonstrated increasing respiratory rate, increase in tidal volume to $+6G_x$, followed by decrease, S-shaped function for minute volume, and minor-image S-shaped function for vital capacity. They observed the occurrence of an oxygen debt during acceleration, less so when breathing oxygen at atmospheric or increased pressure. They considered that acceleration in the $+G_x$ vector interfered mechanically with respiration and demonstrated associated EMG changes. They discuss the possible causes of the oxygen debt.

9. BAKER, A. S., IAKOVLEVA, E. V.
The effect of centripetal acceleration upon sodium and potassium ion levels in urine and saliva.
Voprosy Meditsinfkoi Khimii (Moscow), *6*:615, 1960.
Abstracted in *Aerospace Med.*, *32*:567, 1961.

Sodium and potassium ion levels in urine and saliva before and after repeated exposure to positive acceleration were studied by flame photometry. The sodium ion concentration tended to decrease and potassium to increase under acceleration stress. These changes were more pronounced in individuals with low acceleration tolerance.

10. BARR, P. O.
Hypoxemia in man induced by prolonged $+G_z$ acceleration.
Acta Physiol. Scand., *54*:128, 1962.
WADD–AMRL–TDR–62–137, 1963.

Blackout and cerebral disturbances have been ascribed to adverse effects on the systemic circulation with reduction of cerebral blood flow. Barr et al.[12] (1959) showed that pulmonary circulation is especially susceptible to changes in accelerative vectors and demonstrated that prolonged positive acceleration would produce marked arterial hypoxemia in anesthetized dogs on 100% oxygen. This suggested increased venous admixture.

The author extended the study to man. Eight subjects were exposed to 4 to $5G_z$ wearing a standard anti-G suit. Rate of onset varied from 5 to 25 seconds to peak (in one case 40 seconds). Inspired gas volumes, arterial oxygen saturation, arterial pH, heart rate, and acceleration level were measured. Prolonged exposures consistently caused a drop in arterial oxygen saturation and an increase in respiratory minute volume. Unsaturation proceeded at a rapid pace during the first half-minute and then slowed down, so that the maximum degree was attained before 90 seconds had elapsed. Respiration rate, sometimes after initial apnea, increased during the first half-minute, but showed no greater increase in a prolonged run. Arterial pH showed insignificant changes during exposure. Where the subject breathed 100% oxygen or did not wear an anti-G suit, a limited fall in oxygen saturation occurred. Arterial pH and respiratory minute volume were about the same as with an anti-G suit breathing air. On repeated runs, the fall in arterial oxygen became steeper, increasing with each run. The final level became progressively lower. Hyperventilation became more marked but pH remained essentially unchanged. Oxygen saturation rose sluggishly postacceleration.

Author suggests that venous admixture in the pulmonary circulation (shunting) is the major component in the overall picture. Alveolar-arterial oxygen difference can arise from (1) resistance to diffusion, *i.e.*, diffusion component, (2) shunt component, (3) distribution component. Increased effective weight of blood can produce these by (1) increased intracapillary pressure and formation of edema, (2) congestion with passage of blood through known ventilated alveoli, (3) defective perfusion. This effect is exaggerated by decreased cardiac output. The author suggests that pH remained unchanged because admixed venous blood has a high carbon dioxide content; enlargement of physiological dead space by diminution of total blood flow would impair elimination of carbon dioxide, and presence of acid metabolites from tissues would shift arterial pH in acid direction and counteract alkalosis. In certain other runs arterial oxygen saturation dropped to below 80% at 4½ to 5G.

11. BARR, P. O.
Pulmonary gas exchange in man as effected by prolonged gravitational stress.
Acta Physiol. Scand., *58* (sup. 207), 1963.

The author measured the alterations in ventilation-perfusion relationships during exposure of healthy subjects to $+5G_z$ for 2 minutes while wearing an anti-G suit. Records were obtained of respiratory minute volume, end-tidal carbon dioxide tension, arterial oxygen saturation, and pH, during prerun, run, and postrun conditions. During the run, expired minute volume increased from 8.6 to 20.8 liters per minute and "effective" alveolar ventilation from 4.9 to 9.6 liters per minute (BTPS). The pH increased by 0.03 pH unit from a prerun value of 7.41, and arterial oxygen saturation decreased from 96.2% to 87.4%, indicating a decrease in arterial oxygen tension from 90.1 to 58.0 mm of Hg. End-tidal carbon dioxide tension decreased from 34.5 to 23.9 mm Hg, and arterial carbon dioxide tension from 38.1 to 35.5 mm Hg, indicating an increase of 8.0 mm Hg in the arterial to end-tidal carbon dioxide difference. An increased arterial to end-tidal carbon dioxide difference was thus responsible for the major part of the decrement in end-tidal carbon dioxide tension.

All dead space increased, the alveolar dead space by an average of 170 ml. Calculating on the basis of a decrease in cardiac output to 80% of prerun level, venous admixture increased to 19.8%; with a decrease of cardiac output to 60%, venous admixture would be 15.9%. Prerun value was 5.8%. Oxygen uptake increased from 269 to 410 ml/min, whereas carbon dioxide elimination increased from 216 to 391 ml/min, with a resulting change in respiratory exchange ratio from 0.80 to 0.96.

In the postrun recovery period (8 to 10 minutes after completion of run), arterial oxygen tension showed a residual decrease of 10.9 mm Hg as compared with prerun level. Ninety percent of final change in arterial oxygen tension during the course of a run was completed within 60 seconds. Findings indicate that the equivalent of one-third of the number of alveoli was completely deprived of blood flow. Alveolar ventilation of this portion of the lungs was effectively dead-space ventilation, producing a

large arterial to end-tidal carbon dioxide difference. In dependent parts of the lungs a shunt, representing one-fifth of cardiac output, occurred, mainly from low ventilation-perfusion ratios, but also from impaired diffusion. This results in severe arterial hypoxemia, despite relative alveolar hyperventilation.

12. BARR, P. O., BJURSTEDT, H., COLERIDGE, J. C. G.
Blood gas changes in the anesthetized dog during prolonged exposure to positive radial acceleration.
Acta Physiol. Scand., 47:16–27, 1959. (Authors' Abstract)

Anesthetized dogs were exposed to increased gravitational stress in the head-to-tail direction and arterial oxygen saturation and acid-base balance changes were studied. Simultaneous direct and continuous recordings were made of arterial oxygen saturation and pH as well as respiratory minute volume in centrifuge runs. Application of moderate G forces over several minutes produced severe hypoxemia although 100% oxygen was breathed and hyperventilation was present, indicating a great alveolar-arterial oxygen difference, and accordingly, a large intrapulmonary shunt.

13. BECKMAN, E. L.
Protection afforded the CVS by the CSF under the stress of negative G.
J. Aviat. Med., 20:430, 1949.

The author describes the work done on the cardiovascular system and cerebrospinal fluid (CVS and CSF) aspects of negative G and delineates a carefully conducted series of experiments. Conclusions: CSF and venous pressures measured at the level of the base of the brain in goats varied simultaneously and by approximately the same amounts during exposures ranging from 1.2 to 8.9G_z. Arterial pressures at the same level increased simultaneously with venous and CSF pressures and by comparable magnitudes. The A–V pressure differences developed were found to be not greater than the A–V differences sustained by normal vessels; thus normal intracranial vessels are adequately protected against sudden changes in intravascular pressures by simultaneous changes in CSF pressure. Two explanations of the intracranial hemorrhage reported in the literature are proposed: (a) Torsional stress applied to meningeal vessels by relative motion between brain and meninges produced by rapid acceleration of head; (b) rupture of abnormal intracranial vessels subjected to increased A–V differences.

14. BECKMAN, E. L., DUANE, T. D., COBURN, K. R.
Limitation of ocular motility and pupillary dilatation in humans due to positive acceleration.
AMAL NADC–MA–6–140, 1961.

To determine a satisfactory end point for acceleration stress, 60 subjects were exposed to more than 400 centrifuge runs at varying G_z levels. All subjects demonstrated limitation of ocular motility (LOMA) at between 3.5 and 7G_z, between grayout and blackout. In some the limitation could be overcome by voluntary effort but the resulting movements were ataxic. Neurological examination showed that lower motor neurones to the extraocular muscles are not involved. LOMA is associated with dilatation of the pupils, reaching a maximum at blackout, where pupillary reflexes are absent. A horizontal nystagmus may persist at this stage if previously stimulated by, for example, optokinetic drum. A vertical nystagmus was observed in several subjects during high rates of change of acceleration. When extraocular pressure was applied by skindiver mask, vision was restored, ataxic movements became coordinated, and optokinetic reflexes were restored, but pupils remained partially dilated.

15. BECKMAN, E. L., DUANE, T. D., ZIEGLER, J. E., HUNTER, H. N.
Some observations on human tolerance to accelerative stress.
Phase IV. Human tolerance to high positive G applied at a rate of 5 to 10G per second.
NADC–MA–5302, 1953.

Investigations were made to determine more accurately the voluntary human tolerance to high positive G loads applied at the rate of 5 to 10G/sec. Measurements were made on five adult human males at 15, 12, 10, and 6G, and upon seven subjects at 8G. Subjects were safely exposed to accelerative loads of 15G for durations of 1 to 1.8 sec if the loads were applied at a maximum rate of 8 to 10G/sec. When subjects were exposed to 15G loads applied at 8G/sec, unconsciousness was sometimes produced if the maximum-load duration was greater than 0.5 sec. The visual symptoms of graying, peripheral vision loss, and blackout were not considered to be premonitory signs of impending unconsciousness when the load was 8G or more applied at 7G/sec or more. Unconsciousness produced as a result of loads of 8 to 15G applied at 7 to 9.6G/sec did not produce clinically demonstrable sequelae.

16. BLACK-SCHAFFER, B.
Protection by deep hypothermia and immersion against 2300G acceleration of a non-hibernator (rat) and a hibernator (hamster).
Aerospace Med., 33:286, 1962.

The hamster can be protected against a spin at 2200G of 30 minutes' duration while in deep hypothermia and immersed in a fluid of specific gravity equal to its own at temperatures between 0° and 10° C. The rat can also be protected under the same circumstances but does not permanently survive more than 5 minutes, despite a total period of hypothermia of as little as 50 minutes. Immediate cause of death is a congestive gastroenteropathy believed to be caused by autodigestion of the mucosa by gastric and pancreatic proteolytic enzymes. The author has

found the same lesion in rats after hypothermia without other stress, when hypothermia is more than 70 minutes. He believes that circulatory stasis of hypothermia impairs the normal protective devices of enteric mucosa. Hydrostatic pressure of the spin aggravates the situation. Recirculation of the blood on revival results in absorption of water-soluble substances ordinarily not absorbed, which produce irreversible shock.

17. BLACK-SCHAFFER, B., HENSLEY, G. T., SIMSON, L. R.
Protection of the adult mouse against 1800G acceleration by hypothermic immersion.
Aerospace Med., 32:1119, 1961.

The authors showed that deep hypothermia combined with immersion in a sodium chloride solution of approximately the same density can protect adult mice against 15 minutes of 1800G. This is based on a modification of Newton's law as follows: $F=V(d_1-d_2)a$, where d_1 and d_2 are specific gravities of an immersed homogeneous body and the immersion fluid, V is volume of body, and a is acceleration. If d_1 equals d_2, the force exerted on the body within the system is zero. Since a mouse is an obligate nasal breather, obstruction of the small-caliber nasal passage by blood and mucus plugs causes death after revival. Despite this, one mouse recovered and survived for 15 months after an 1800G spin of 10 minutes' duration.

18. BONDURANT, S.
Effect of acceleration on pulmonary compliance.
Federation Proc., 17 (Suppl 2) :18, 1958.

By the method of Mead and Wittenberger, the author measured pulmonary compliance in five subjects during acceleration of 30 seconds' duration on the human centrifuge. Observations were made at $+3G_z$, $+3.5G_z$, $+4G_z$, and $+5G_z$. The mean of five measurements at each G level was determined. Compliance decreased from a prerun group mean of 0.167 ± 0.026 liters per cm of water to 0.121 ± 0.022 at $3G_z$, and 2.106 ± 0.011 at $3.5G_z$. During transverse acceleration, compliance decreased from a prerun value of 0.174 ± 0.038 liters per cm of water to 0.105 ± 0.031 at $4G_x$ and to 0.083 ± 0.041 at $5G_x$. Changes from control values are all significant at level of P less than 0.05. During $+G_x$ acceleration, the functional residual capacity increased by 100 to 600 cc. During $+G_x$, increase was less than 250 cc. Such changes are not usually associated with a change in the compliance. Both positive and transverse acceleration, therefore, cause an apparent decrease in compliance not related to change in FRC.

19. BONDURANT, S., CLARKE, N. P., BLANCHARD, W. G., MILLER, H., HESSBERG, R. R., HIATT, E. P.
Human tolerance to some of the accelerations anticipated in space flight.
U. S. Armed Forces Med. J., 9:1093, 1958.
WADC TR–58–156.

This paper represents a summation of the work of the authors and others in this field. Since new evidence was found of marked decreased tolerance from intermittent breaks in acceleration pattern, the plateau acceleration pattern was commonly used with rates of onset of 0.1 to 8G per second. In some studies three-stage rocket simulation was used, with peaks of 8, 10, or 12G and onsets of 1G per 12 sec, 1G per 7 sec, and 1G per 4.5 sec, respectively. Valuable diagrams of tolerance and effects of position are included.

Transverse G. Tolerance in the conventional seated position is limited at 8G by dyspnea and chest pain. If angle of trunk relative to direction of acceleration is greater than 70°, severe chest pain limits tolerance at 7G. Below 70° there is more longitudinal application of inertial force, with resulting blackout at lower G. Best tolerance is with subject leaning in direction of acceleration at 65° to 70°. With legs flexed or body semisupine, blackout limits tolerance above 8G. Tolerance is greater seated at 65° to 70° angle than semisupine, probably from greater displacement of blood from legs. Seated at optimum angle, and semisupine, respiration is difficult above 4G and may be limiting at 6 to 8G. Above 6G, petechiae of back and antecubital fossae occur regularly. After acceleration in these positions disability is variable—unsteady gait, dizziness, vertigo, and occasional nausea from 1 to 5 minutes. Acceleration in the $-G_z$ vector is limited by the restraint system and by head position, which must be vertical. With extended legs, calf and thigh pain limits tolerance to 5G and may persist for several days. In optimal position, $-G_x$ tolerance is limited at 8G.

Positive G. Tolerance without G suits is approximately 4G for 20 minutes or 3G for 60 minutes. Petechiae commonly occur after 10 minutes above 3.5G. Tolerance is limited below 4G by fatigue, backache, and headache. With slow rate of onset (0.1G/sec) blackout is rare below 4G. Occasional episodes of syncope occur. Residual effect is variable and may be prostrating for hours, particularly weakness and dizziness.

Rates of onset. With transverse acceleration, rates of onset faster than those described have produced slightly longer (seconds) tolerances at high G. Slower rates have shortened tolerances. Similar effect was found with positive acceleration. Note that 15G for 5 seconds semisupine is tolerable with rate of onset of 6G per second and that during World War II two subjects tolerated 17G for 1 minute at a rate of onset of 1G for 6 seconds.

Conclusion. The optimal position appears to be seated with 20° inclination of trunk toward acceleration, legs fully flexed (seated facing forward). Three-stage accelerations peaking at 8, 10, and 12G are tolerable in this position. In a semisupine posi-

tion, peaks to 10G are tolerable. Accelerations less than 4G are tolerable in all positions.

20. BRENT, H. P., CAREY, T. M., POWELL, T. J., SCOTT, J. W., TAYLOR, W. J. R., FRANKS, W. R.
Synergism between effects of hyperventilation, hypoglycemia and positive acceleration.
Aerospace Med., 31:101, 1960 (Authors' Summary)

Eighteen medically fit aircrew officers were exposed to combinations of voluntary hyperventilation, 3.4G for 5 seconds on the centrifuge, and changes in glycemia 1 hour and 3 hours after glucose feeding. A large proportion of the subjects were incapacitated by the effect of two or more of the combined stimuli, although these same stimuli, when acting separately, had failed to induce comparable disturbances in cardiac or cerebral function. The experimental findings are summarized as follows:

1. Voluntary hyperventilation plus 3.4G, 1 hour after glucose, produced EEG slow activity in seven subjects and physical signs indicative of complete incapacitation in six of these. A wide range of signs and symptoms appeared in the remaining ten subjects. Five of the EEG disturbances occurred at above-fasting blood glucose levels.

2. Voluntary hyperventilation plus 3.4G, 3 hours after glucose, produced EEG slow activity in 12 of 17 cases, with physical evidence of complete incapacitation in 8 of these. A large majority of the EEG disturbances (11 of 12) occurred in the presence of a fasting or lower-than-fasting blood glucose level.

3. Voluntary hyperventilation induced an increase in heart rate when combined with the positive acceleration. They produced a greater increase and gave rise to more cardiac irregularities than had appeared with either condition acting alone.

4. Electroencephalographic and electrocardiographic disturbances tended to occur together, and both were commoner at fasting or lower-than-fasting blood glucose levels than at higher levels.

5. Of the 17 subjects who underwent all phases of the experiments, 16 had developed fasting or below-fasting blood glucose levels at 3 hours after their glucose meal.

21. BRISSENDEN, R. F.
Some ground measurements of the forces applied by pilots to a side located controller.
NACA TN 4171, 1957.

22. BRITTON, S. W., PERTZOFF, V. A., FRENCH, C. R., KLINE, R. F.
Circulatory and cerebral changes and protective aids during exposure to acceleratory forces.
Am. J. Physiol., 150:7, 1947.

The authors exposed large numbers of monkeys, dogs, and cats to accelerations up to as much as 50G for varying durations. They observed an increase in heart rate (5 to 30 per minute) on exposure to positive G, proportional to the time and intensity of acceleration. Exposure to 3 to $4G_z$ for 5 to 10 minutes resulted in bradycardia and irregularity, with rapid recovery on stopping acceleration. Acceleration in the $-G_z$ vector produced no significant difference in the heart rate of the dog. EGG showed a marked drop in voltage over the left precordium during $+G_z$ exposure, with a simultaneous increase over the right precordium. Alteration of the heart position by direct traction, and operative variation of blood flow through the heart, showed that these ECG changes were produced largely by the position of the heart and partly by the amount of blood flowing through the heart. Carotid blood pressure fell sharply under $+G_z$ acceleration, with a concomitant rise in the femoral pressure. Pressure measured in a hydrodynamic model showed similar changes. Arterial pressure was found to be influenced by a carotid sinus reflex about 6 to 8 seconds after starting exposure to acceleration. Reduction in blood flow occurred in the carotid artery within a few seconds after starting acceleration and was proportional to the acceleration applied. At $+3G_z$ to $+4G_z$ for 10 seconds, flow in the carotid or brachial vessel usually reached zero. Acceleration of 4 to $8G_y$ produced moderate increases in the heart rate and in carotid pressure and flow.

Delta waves appeared in the EEG of 75% of monkeys tested within a few seconds of acceleration. These sometimes disappeared toward the end of a 10-second run and appeared again when the centrifuge stopped. Protection against acceleration was afforded by a belt or a cuff around the abdominal area inflated to about 300 mm Hg pressure. The advantage afforded by the belt varied from 1 to 3G. Injection of adrenalin gave moderate and brief protection.

23. BROWN, G. E., JR., WOOD, E. H., LAMBERT, E. H.
Effects of tetraethyl ammonium chloride on cardiovascular reactions in man to changes in posture and exposure to centrifugal force.
J. Appl. Physiol., 2:117–136, 1949.
Abstracted in STL–9990–6302–KU–000.

Studies were carried out on the effect of the drug on the reactions produced in the cardiovascular system by tilting from the supine to the upright position, exposure to centrifugal force, and by the sitting posture.

24. BROWN, J. L.
Acceleration and Human Performance.
In H. W. Sinaiko (ed.), *Selected Papers on Human Factors in the Design and Use of Control Systems*, Dover Pub., Inc., New York, 1961.

This is a comprehensive review of the effect of sustained acceleration on performance. Performance decrement is related to (a) effects on senses, (b)

mechanical effects, and (c) central effects. Topics include vision, vestibular, and kinesthetic senses; ability to move and apply forces; ability to undertake complex tasks and control of vehicles; and higher intellectual function.

25. BROWN, J. L., BURKE, R. E.
Effect of positive acceleration on visual reaction time.
NADC–MA–5712, 1957. (Authors' Summary)

Reaction time to visual test signals was measured for two subjects during exposure to positive acceleration. Two test light luminances, 4560 millilamberts and 0.025 millilambert, and two regions of the retina, one close to the foveal center of the eye and the other far removed from foveal, were investigated. When measured in terms of a visual effect, acceleration tolerance was higher for the brighter light. It was also higher when the more central region of the retina was stimulated. Reaction time is increased significantly with increases in positive acceleration below the tolerance level. Increase in reaction time does not occur until after a minimum of about 5 seconds' exposure to acceleration between 3 and 6G.

26. BROWN, J. L., LECHNER, M.
A survey of research.
NADC–MA–5503, Bureau of Med. and Surg., AMCL, USNADC, Johnsville, Pa., March 22, 1955.

This is a comprehensive review of the literature on man's performance under sustained acceleration. The authors point out that there are a number of serious gaps in our knowledge of even the most elementary aspects of sensory function and the mechanics of motor performance as these are related to acceleration. Virtually no serious attempt has been made to investigate the effects of acceleration on complex functions involving higher mental processes. The apparent lack of adequate information regarding exactly what performance may be required of a pilot exposed to acceleration is a serious problem. Such information must be obtained before basic data can be usefully employed.

27. BROWNE, M. K., FITZSIMONS, J. T.
Electrocardiographic changes during positive acceleration.
FPRC–1009, 1957. (AD–141–045)

The authors analyzed pulse rates, pulse rate intervals, and changes in the electrical axis in 53 subjects during 366 runs under positive accelerations of 3 to 5G. They used standard leads, 1, 11, and 111, and unipolar leads aVL, aVR, aVF, and V_1 to V_6. Small silver electrodes were attached to the skin. Vector cardiograms were obtained in some subjects by a method of derivation using a rectangular coordinate system, and in others by presenting the loop on an oscilloscope and photographing it. Respiration was recorded by a thermocouple. Centrifuge runs were at 3, 3.5, and 4G for 15 seconds, allowing 4.5 seconds from 2G to peak G. Two groups of subjects were used, experienced and inexperienced.

Pulse rates. Inexperienced subjects showed higher pulse rate at 3G, statistically significant at the 1% level, but not statistically significant at 3.5 and 4G. Time to reach maximum pulse rate was significantly longer ($P=0.02$) at 3G but insignificant at the higher levels. The PR interval grew progressively shorter with increase in pulse rate.

ECG. Apart from change in the electrical axis, no characteristic ECG abnormalities were observed, except possibly on one occasion when consciousness was lost. In all subjects except two with horizontal hearts, under G the axis became more vertical in the frontal plane, more vertical in the sagittal plane, and showed anticlockwise rotation in the horizontal plane when rotation occurred. In the two horizontal cases, the axis became more horizontal in the frontal and sagittal planes, while one rotated clockwise and the other showed no rotation. No relationship could be observed between the electrical and anatomical axes as obtained by X-ray.

Vectorcardiograms. These represent the changing electrical axis. Although the direct method was found more valuable than the indirect, the technique was of little use except in subjects normally having horizontal hearts at rest.

Increase in pulse rate was believed partly due to carotid sinus effects and partly to an adrenal medullary response to stress. The latter cause is indicated by the significant difference in pulse rate reached by experienced subjects and inexperienced subjects. If fall in blood pressure were the only factor there would be no such difference. At higher G levels, where maximum response is required on hemodynamic grounds, the difference tends to disappear though inexperienced subjects tend to reach maximum value sooner, suggesting that apprehension is still important. Wearing of a G suit caused no greater percentage increase in pulse rate, though the time taken to reach maximum was decreased, probably from maintenance of adequate venous return. This is in contrast to findings of Wood and Lambert [190] (1952), perhaps because of different G suit, or perhaps because of different conditions.

Changes in electrical axis were greater with respiration than with acceleration. Respiration under G has an even greater effect on ECG than at rest.

Although T-wave changes occurred, they were no greater than T-wave changes with respiration, both at rest and under G. If, however, maximum positional changes have occurred and further progressive changes occur, these probably represent ventricular strain and an important sign of impending unconsciousness.

28. BÜHRLEN, L.
An investigation of the sense of orientation while the human body is subjected to centrifugal force (Versuche uber die Bedeutung

der Richtung beim Einwirken von Fliehkraften auf den Menschlichen Körper).
Luftfahrtmedizin, v. *1*, 1937.

29. BURMEISTER, H.
Untersuchungen uber Anderungen der optischen Reaktionszeit des Menschen beim Einwirken höher Fliehkräfte.
Luftfahrtmedizin, *3*:277–284, 1939.

30. BURTON, A. C.
Hemodynamics and the Physics of the Circulation.
In T. C. Ruch and J. F. Fulton (eds.), *Medical Physiology and Biophysics*, W. B. Saunders Co., Philadelphia and London, 1960.

31. BURTON, R. R., RICHARDS, W. P. C., SMITH, A. H.
Pathology of chronic acceleration.
Presented at the 34th Annual Meeting of the Aerospace Medical Association, 1963.
Abstracted in *Aerospace Med.*, *34*:249, 1963.

Chronic acceleration produced by centrifugation is a highly lethal treatment. When growing chickens are exposed to an accelerative force of 2.5G, about half of them die in 11 days—however, the mortality curve is not a smooth exponential relationship. Resistance to chronic acceleration requires a true physiological adaptation, and the factors involved in the adaptation are heritable. After five generations of selection (on the basis of survival of chronic acceleration), the exposure to 2.5G leading to a 50 percent mortality is increased to 60 days.

When animals dying during chronic acceleration are examined postmortem a variety of pathological changes are evident, but none is present systematically. It seems likely that several pathologies are involved in chronic-acceleration death. Recent routine examination of centrifuged birds has indicated about 25 externally observable items which change in some birds during chronic acceleration, including general appearance, evidence of normal nutrition, posture and locomotion, and reflexes. When these were graded on a plus (normal), or minus (abnormal) basis a score varying from +20 to −20 was obtained. On the basis of rate of development of symptoms (zero-time being the onset of symptoms, rather than start of centrifugation) the birds can be divided into five groups.

32. CANFIELD, A. A., COMREY, A. L., WILSON, R. C., ZIMMERMAN, W. S.
The effect of increased positive radial acceleration upon discrimination reaction time.
J. Exp. Psychol., *40*:733, 1950.
(Abstract from STL–9990–6302–KU–000)

Twenty-three male college students served as subjects. All experimental trials were given with subject wearing an anti-G suit. The problem was to determine the direction that a red light lay from a green one on an exposure panel and then to make a simple motor response indicating that direction as quickly as possible. A series of four experimental days were given each subject. On each of the four days subjects were tested twice at the normal resting condition (1G) and twice each at positive radial accelerative forces equal to 3 and 5G.

33. CANFIELD, A. A., COMREY, A. L., WILSON, R. C.
A study of reaction time to light and sound as related to positive radial acceleration.
J. Aviat. Med., *20*:350, 1949. (Authors' Summary)

It is concluded on the basis of the experiment reported here that the reaction time to both light and sound stimuli becomes significantly longer under conditions of increased radial acceleration. The superiority of reaction to sound, in addition to the known failure of sight at crucial G-levels, suggests the advisability of presenting important signals to the pilot in the auditory sense modality rather than the visual. This is especially vital in those cases where such signals demand emergency action. The tremendous speed of the aircraft of today and tomorrow will necessitate action to conserve every split second. Taking steps such as those suggested here should help to take some of the burden from the limiting factor in aircraft design—the pilot.

34. CHAMBERS, R. M.
Control performance under acceleration with side-arm attitude controllers.
NADC–MA–6110, 1961.

Twenty-four pilots trained in simulator centrifuge operation served as subjects. They were exposed to forward ($+G_x$) accelerations peaking at 1, 3, 6, 9, 12, and 15G, representing the first one and a half stages of a four-stage launch profile, for a total duration of 120 seconds, while restrained at head, chest, and legs in a contour couch, and controlling a closed-loop system.

Among results were reports of experience as follows: At 3G, slight difficulty in focusing and slight disorientation, both subsiding with practice. At 6G, tightness in chest, mild chest pain, some loss of peripheral vision, some difficulty in breathing and speaking, decrease in depth of visual field, additional effort in scanning and maintaining focus, blurring of instruments, and tendency to overcontrol. At 9G, chest pains, sensation of heaviness in chest and stomach, difficult breathing, requiring tensing chest and stomach, slight blurring, tendency for eyelids to close over eyeballs, reduced peripheral vision, occasional tears, tunneling of vision, greater concentration and more effort required in control, loss of "feel," tendency to make inadvertent control inputs, and hesitation to make control inputs because of possibility of inadvertent control. At 12G, severe difficulty in breathing, severe pressure and discomfort on chest, chest pain, physical fatigue, dimness, loss of peripheral vision and visual acuity with tearing;

scanning almost impossible, control difficult and great concentration required. At 15G, some reported complete recurrent impairment of vision, extreme difficulty in breathing and speaking, loss of sense of feel, difficult control; visual symptoms were improved by oxygen and alteration in back angle.

Mean estimates of performance impairment of six pilots were 22.5% at 6G, 32.5% at 9G, and 52.5% at 12G, as compared with static baseline. For two pilots the impairment estimation at 15G was 72.5%. Within the physiological tolerance limits, performance during centrifuge runs was not greatly reduced as compared with performance on the same problems statistically, though more work, more concentration, and more effort were required.

35. CHAMBERS, R. M.
Long term acceleration and centrifuge simulation studies.
Aviation Medical Acceleration Laboratory, 1963.

A useful review containing good material on acceleration nomenclature, and also visual performance. Summarizes much of the work carried out at AMAL in contour-couch G protection, dynamic flight simulation, effects of positive-pressure breathing, discrimination, and complex skill and higher mental functioning.

36. CHAMBERS, R. M., HITCHCOCK, L.
The effects of acceleration on pilot performance.
NADC–MA–6219, 1963.

Physiological tolerance limits define the maximal end points for safe exposure of a particular physiological system to stress. Performance tolerance limits define the upper limits of reliable functioning of a particular performance-ability system. Beyond the tolerance limit, performance deteriorates extremely rapidly.

Vision. The relationship, with respect to vision, between amplitude of G and duration at peak G has not been established. As G increases, a given level of visual acuity may be maintained by increasing the size of the target or the amount of luminance. Thus, the ability of pilots to read instruments under acceleration is partially a function of the level of illumination, and of the contrast between figure and background as background luminance is decreased. The available oxygen under acceleration conditions affects discrimination. At $+3G_x$, $+4G_x$, and $+5G_x$ levels, positive pressure plus 100% oxygen provides conditions where less visual contrast is required than under other similar experimental conditions. Similar results were found for transverse G and for 100% oxygen without positive-pressure breathing.

Discrimination reaction time. Acceleration modifies the ability to respond to stimuli. Thus, the response time in discrimination of colored lights is longer under G_x acceleration than under normal conditions. The study by the authors of 5-minute exposures to $6G_x$ showed that in the first block of 25 trials the response scores were slower than average; during the second series of trials response scores were still slower, but during the third series performance significantly improved. This suggests that subjects had learned to maintain their physiology and performance under acceleration stress and indicates that learning how to perform during exposure to stress is a primary factor in determining performance ability. A similar increase in response time under G_x exposures is observed with auditory stimuli.

Complex psychomotor performance. Tracking deteriorates under transverse acceleration but rapidly returns to normal as acceleration returns to normal. A similar test carried out after 12 hours of submersion in water shows the same decrement. Tracking tasks have been carried out under simulated reentry acceleration. Tracking performance was impaired at high G levels, but pilots were able to maintain proficiency above the minimum levels considered necessary to continue the run. The outstanding problems at 12 and $15G_x$ levels were impairments in vision, difficulty in breathing, and difficulty in operating the control device. In another reentry study, pilots indicated they were unable to concentrate on more than one or two things at the same time at high G. Thus, they found it necessary to neglect some parts of their tasks. Although only normal physical effort was required to perform the task under low acceleration, 100% effort was required at the highest acceleration, $15G_x$. At levels below $7G_x$ there appeared to be little effect of the acceleration on the control task.

Higher mental abilities. No conclusive information is available regarding the effects of acceleration on the basic intellectual abilities required for functions such as immediate memory and the ability to process information. In the authors' study, where subjects, during acceleration of $5G_x$ for 5 min, were required to compare a symbol presentation with the memory of a symbol which had been presented two, three, or four presentations previously, there was an increase in the latent period between presentation and response. Subjects reported that their performance deteriorated under G and that the exposure was extremely stressful. In some studies discrimination time was impaired not only under G but also for some time after termination of G. In a complex matching task the results suggested that proficiency in immediate memory was maintained at least through $+5G_x$, but some impairment was observed at $+7G_x$ and $+9G_x$. In prolonged exposure, for example at $+2G_x$ for 4 hours, no significant impairment in matching memory tasks was observed.

Specific mission tasks. Three general categories of acceleration effects were noted during acceleration runs simulating orbital missions of the Mercury type: (1) Insertion of specific control inputs of which the pilots were often unaware, (2) disruption

of the timing and precision of pilot control, (3) discrete task function such as an operation override. These were affected by accelerations which preceded and/or followed them, though the operations themselves were performed under minimal acceleration loads, and could result in excessive fuel utilization. The general control effects may occur without knowledge of the pilot and are chiefly in the pitch and yaw planes. The roll axis is not significantly affected.

Discrete task responses. The overall mean response time to tally panel indications, that is, warning indications represented by a panel light, is not affected by acceleration, but under dynamic conditions there is increased response variability. It may also be noted that individual pilots respond differently under acceleration stresses. Panel indication may be "red light" or "no light." Response time was considerably longer when "no" indication was given than when the red light indication was given. On very few occasions did the pilots fail to make any response that would indicate recognition of panel indication. Average response times were not significantly affected by simulation of altitude (5 psi). Although not significant, an actual increase in response time was observed at altitude.

37. CHAMBERS, R. M., NELSON, J. F.
Pilot biomedical and psychological instrumentation for monitoring performance during centrifuge simulations of space flight.
NADC–MA–6308, 1963. (Authors' Summary)

This report represents some of the results of recent centrifuge acceleration research and training projects in which the biomedical, psychophysiological, and psychological performances of pilots were monitored and measured. Monitoring and recording instrumentation techniques are described, and an attempt is made to identify and quantify some of the capabilities and limitations of pilot performance during exposure to accelerations which vary in magnitude, duration, direction, rate of onset, and profile complexity. Apparatus and methods are presented and discussed for monitoring visual disturbance, discrimination and response behavior, and complex skill behavior, and an approach is made to the problem of monitoring higher mental functioning. The pilots and other volunteers in these training and research programs were the 7 Mercury astronauts, 6 Dyna-Soar consultant pilots, approximately 35 other test pilots, and approximately 40 other military and civilian volunteers.

38. CHERNIACK, N. S., HYDE, A. S., WATSON, J. F., ZECHMAN, F. W.
Some aspects of respiratory physiology during forward acceleration.
Aerospace Med., *23*:113–120, 1961.
(Abstract from STL–9990–6302–KU–000)

A review of current experiments in respiratory physiology during forward acceleration is presented. Dyspnea, inspiratory chest pain, tracheal tugging, paroxysmal coughing, and a sensation of weight on the thorax are typical symptoms occurring during forward acceleration. Fine crepitant rales are sometimes heard over the posterior lung fields for several minutes after centrifugation if the acceleration has been prolonged and has been of considerable magnitude.

Hemoptysis lasting 6 hours has occurred on one occasion. There is, naturally, considerable variation among subjects and even in the symptoms experienced by the same subject from day to day.

No serious cardiac aberrations were noted during several experiments with levels of forward acceleration up to 16.5G and with duration up to 3 minutes at 12G.

39. CHRISTY, R. L.
Effects of Radial and Angular Accelerations.
In H. G. Armstrong (ed.), *Aerospace Medicine*, ch. 16, The Williams and Wilkins Co., Baltimore, 1961.

40. CLARK, C. C.
Observations of a human experiencing 2G for 24 hours.
Paper presented at 31st Annual Meeting of the Aerospace Medical Association, 1960.
(Abstract from STL–9990–6302–KU–000)

A subject rode the Navy's Johnsville human centrifuge for 24 hours at 10 rpm of the 50-foot-radius arm, giving a resultant linear acceleration of 2G perpendicular to the floor of the gondola, which was positioned at an angle of 60° with respect to the centrifuge arm. The subject rode, for the most part, in a reclining chair with a back angle of 45° with respect to the gondola floor. Experiments were made with the rotational illusions generated by head motion while on the turning centrifuge. These illusions had a threshold with a head angular velocity of about 0.06 rad/sec. Head motions of about ten times this rate were nauseating. The subject stood up and walked a few steps 2 hours after starting, but had a dimming of the peripheral vision and some nausea when attempting this 12 and 22 hours after starting.

The subject cooked, ate, slept, about 5 hours (subject's estimate), measured his pulse rate, blood pressure, temperature, and time-interval estimation accuracy, and looked for handwriting and speech changes. Abdominal discomfort for the first 2 hours and a mild frontal headache were relieved by aspirin. Two additional tablets were taken during the run. Sixteen hours after the start, an anesthesia sensation developed in the ring and little fingers of the left hand, and some tingling sensation remained for about 2 months after the experiment. Water (and milk) input during the experiment was 2250 cc; urine output was 890 cc. During the experiment the white blood count increased from 11,300 to 22,000 per cubic millimeter. Symphony music when desired provided the subject with what he considered a very important

distraction from generalized discomforts, particularly of areas in contact with the chair. A feeling of lightness lasted only about 30 minutes after the centrifuge stopped. An abrupt head motion 30 minutes after the run produced retching, but other recovery seemed uneventful.

41. CLARK, C. C., BLECHSCHMIDT, C., GORDON, F.
Pilot Compartment Airbag Restraint Program.
Martin Rep. ER 13551, 1964.

42. CLARK, C. C.
Some body displacements and medical effects of lateral accelerations during Navy centrifuge simulation of ejection capabilities from the Army AO aircraft.
NADC–MA–6044, 1961.

To investigate problems of ejection under lateral acceleration, the author exposed subjects to G_y acceleration, one subject from 1 to 3G, three subjects from 1 to 4G, and one of the latter to 5G. Onset was 12.5 seconds to peak, plateau was 10 seconds, and offset 6 seconds. A total of 18 runs was made. Displacement occurred at shoulder, hip, and head, the shoulder displacement chiefly affecting the right clavicle with bruises lasting for several days. Petechiae were seen about right orbit and right temple at $-5G_y$. Petechiae are expected when local capillary pressures exceed the usual values by 100 to 150 mm Hg for a few minutes. This is equivalent to a blood column of 120 to 200 G-centimeters. Scleral hemorrage occurred at $-4G_y$, along with flushing of right side of face, subjective feeling of warmth on the right, and increase of sweat on the right. Headache persisted later. Hemorrhage disappeared after 2 weeks. Oculogravic illusion occurred markedly in one subject, the nature of the sensation varying with the intensity of the G. There was no significant change in respiration, including vital capacity. Blurred vision occurred in one subject for 15 minutes postrun. The cause is unknown, but possibly related to deformation of globe relative to cornea.

43. CLARK, C. C., AUGERSON, W.
Human acceleration tolerance while breathing 100% oxygen at 5 psia pressure.
Paper presented at 32nd annual meeting of the Aerospace Medical Association, Apr. 1961. (Authors' Summary)

Low-pressure gas provides less support against collapse of the chest under acceleration than gas at atmospheric pressure. With pure oxygen, collapsed lung structure may not reinflate as rapidly after acceleration. Low-pressure studies indicate individual variation in response to $+G_x$, or $+G_x$ tumbling to $-G_x$, ranging from severe chest pains to temporary post-run dyspnea, to no unusual sensation but with minor atelectasis detectable by X-ray or moderate post-run reduction in vital capacity, to no detectable responses at all. Injury may be more severe at lower G for longer duration, with normal respiration, than at higher G for shorter duration with strain, including valsalva maneuver with inflated chest above $+8G_x$. With training in breathing and straining, no subject was incapacitated.

44. CLARK, C. C., GRAY, R. F., HARDY, J. D., SMITH, F. K.
A discussion of restraint and protection of the human experiencing the smooth and oscillating acceleration of proposed space vehicles.
NADC–MA–5914, 1959.

This paper initially discusses acceleration terminology, space-flight simulation, and acceleration problems associated with the X-15 and several proposed space vehicles. Authors also consider means of protection by restraint and contour couches. Considerations of water immersion are examined, and high-G protection using the G capsule is presented in detail with appropriate records.

45. CLARK, C. C., HARDY, J. D., CROSBIE, R. J.
Some flight and flight acceleration land marks.
In Human Acceleration Studies, NAS–NRC Pub. 913, 1961.

The following are extracts: 1903: A. P. Thurston was thrown to the floor and went unconscious at 6.4G in a Maxim flying machine restrained by wires. Probably first human to become unconscious under acceleration. 1911: Zahm suggested an accelerometer design, and tested factors of safety of wings by static loading and the wire tensiometer. 1911, Dec. 31: John Moisant was killed when thrown from his aircraft abruptly steepening a dive, without lap belt, possibly due to centrifugal forces. 1912: Wilford Parke, in a spiral dive, was thrown outboard, but caught wires and landed safely. 1917: G. F. C. Searle built and flew with a recording accelerometer showing 4.2G in a dog fight maneuver. 1918: Major V. H. reported that "the sky appeared to be so gray" and he fainted, while doing a tight turn at 4.6G. 1922: Lt. K. L. Maughan in the Pulitzer race reported going unconscious on the turns. 1923: Lt. Al Williams, winner of the Pulitzer race at a speed of 243.7 mph, said that in practice flight he passed out completely on a turn. 1924: James Doolittle described maneuvers at 7.8G carried out very abruptly without trouble, but at 4.5G for a few seconds producing loss of vision. 1927: Luke Christofer made abrupt airplane pull-ups, reaching 10.5G. 1930: Schneider Trophy race pilots reported the occurrence of blackout on turns. 1935: Large German centrifuge at the Sanitats-Versuchsstelle der Luftwaffe. 1940: RCAF centrifuge put into operation in Toronto. 1942: Mayo Clinic centrifuge put into operation. 1945: Pensacola centrifuge put into operation.

46. CLARK, C. C., HARDY, J. D., CROSBIE, R. J.
A proposed physiological acceleration terminology with historical review. IV. The use of G or g.

In Human Acceleration Studies, NAS–NRC Pub. 913, 1961.

The authors discuss the various terminologies for use of the term G. They propose that G be used for the unit vector of acceleration normalized to the gravitational constant g_o and representing the full reactive load, including both displacement and gravitational aspects. They suggest that g will continue to be used for displacement acceleration. Thus, in straight and level flight at constant speed, an aircraft has $a_z=0_g$, but pilot experiences $1G_z$. Similarly an aircraft looping with $a_z=1_g$ would expose the pilot to $2G_z$ at the bottom of the loop and 0^G at the top.

47. CLARKE, N. P., BONDURANT, S., LEVERETT, S. D.
Human tolerance to prolonged forward and backward acceleration.
WADC–TR–58–267, 1958.
(Abstract from STL–9990–6302–KU–000)

Optimal body positions for forward and backward acceleration are defined. Plateaus between 2 and 12G were maintained with optimal positioning, until subjective loss of a critical faculty occurred. Forward acceleration of the seated subject with trunk inclined 25° in the direction of acceleration was limited above +10G by blackout and below +10G by inspiration dyspnea and substernal pain. Using a specially designed restraint suit, backward acceleration of the seated subject with trunk and head erect was limited above −10G by discomfort of restraint and increases in vascular pressure in the legs, and below −10G by these factors as well as dyspnea. Three-stage rocket-like profiles of forward acceleration, calculated to give orbital velocity, were found tolerable by selected subjects at peak accelerations of +8G, +10G, and +12G.

48. CLARKE, N. P., HYDE, A. S., CHERNIACK, N. S., LINDBERG, E. F.
A preliminary report of human response to rearward facing re-entry accelerations.
WADD–TN–59–109, 1959.

To explore accelerations expected in the reentry of a nonlifting body from orbit the authors exposed seven subjects to an acceleration profile of 0.14G per second to 8.5G, then 0.32G per second to 16.5G, with a mirror-image decay. In some experiments subjects wore an MC–2 full-pressure suit pressurized to 3 psi. Performance was measured on an ILS type of tracking task. Velocity of airflow was measured continuously by a pneumotach from which tidal volumes were determined. ECG was recorded. Results showed a systematic reduction in tidal volume and increase in respiratory rate. Blackout was approached in 3 of 12 experiments. The two subjects with pressure suits subjectively felt that the uninflated suit did not impair tolerance, and with the suit pressurized at 3 psi two subjects attained a peak of 16.5G. Data from the tracking task were variable and appear to depend on degree of learning. Decrement occurred with peak G. Failure of tolerance was due to chest pain and dyspnea. It is suggested that the use of a G-suit improves vision above 12G.

49. COCHRAN, L. B., GARD, P. B., NORSWORTHY, M. E.
Variation in human G tolerance to positive acceleration.
Rept. 001–059.02.10, USN, SAM, 1954. (Authors' Abstract)

Human G tolerance and some of the variations to positive acceleration were determined on 1,000 individuals, composed of the following groups: (1) Master control, (2) naval aviators, (3) referral students, (4) naval aviation cadets, and (5) miscellaneous. The differences in mean G tolerances for all groups tested for loss of peripheral vision, blackout, and unconsciousness were not significant. Also a great range in human G tolerances for these symptoms was determined. Neither the time required to attain "peak G" nor the G level had any significant effect on the time required to produce these symptoms in individuals at various G stresses.

50. CODE, C. F., WOOD, E. H., LAMBERT, E. H.
The limiting effect of centripetal acceleration on man's ability to move.
J. Aerospace Sci., *14*:117–123, 1947.
NAS–NRC–CAM–436, May 1945.

51. CODE, C. F., WOOD, E. H., STURM, R. E., LAMBERT, E. H.
The sequence of physiological events in man during exposures to positive acceleration.
Federation Proc., *4*:14, 1945.

There is a definite sequence to the physiologic events that occur in the comfortably seated human being during exposure to positive acceleration. This sequence is divided into two distinct periods: the period of progressive failure and the period of compensation. During the period of progressive failure, the pulse rate progressively increases; the amount of blood in the ear may be gradually reduced or abruptly lost; the blood pressure at the level of the base of the brain declines; and reductions of vision and consciousness may occur. As accelerations of greater intensity are experienced, the extent of these changes is increased.

The period of progressive failure is usually terminated by compensatory reactions, which become effective about 6 to 11 seconds after the onset of acceleration. During the period of compensation the blood pressure rises, the ear pulse may return or increase, the amount of blood in the ear pulse may return or increase, the pulse rate increase is checked, and the pulse may slow. If these compensatory changes are sufficiently effective, recovery from symptoms (loss of vision and consciousness) will occur.

52. COLLINS, C. C., CROSBIE, R. J., GRAY, R. F.
Letter report concerning pilot performance and tolerance study of orbital re-entry acceleration.
NADC–LR–64, 1958.

This report concerns a preliminary study of human tolerance to the reentry accelerations expected in zero-lift vehicles. The first phase of the study involved expected reentry G time histories to a peak of 12G, and the second phase to 20G. The AMAL centrifuge was used, with the subjects riding in a semisupine position on a molded contour couch. All subjects wore standard Navy anti-G suits and strained during runs. The G-load was represented by a haversine pattern during which, at peak, the subject was exposed to more than 20G for approximately 5 seconds. Two subjects rode to 20G; one of these blacked out with a back angle 17° from the horizontal. By adjusting the angle to 10° from horizontal, the subject continued to 20G without blackout.

No chest pain was reported although breathing became difficult at high G levels. Blurring of vision was reported at higher G levels. Subjects could operate a small wrist control stick and a thumb switch. Visual tracking showed decrement, but improved with continued exposure. No disorientation was encountered during runs, but first movement of the head after runs produced disorientation.

53. COLLINS, C. C., GRAY, R. F.
Letter report concerning pilot performance and tolerance studies of re-entry acceleration.
NADC–LR–90, 1959.

This investigation was part of a series of studies to evaluate molded contour couches. Using the AMAL centrifuge, with the subject in a semisupine position on a NASA molded contour couch, subjects were exposed to haversine G loads. With a back angle of 17°, peaks increased progressively to 20G. At 20G, blackout occurred, establishing the limit for 17° back angle. No pain was experienced during these runs. With a back angle of 14°, limit was established by blackout at 23G. No pain was experienced. With a back angle of 8°, subject was able to withstand peak acceleration of 25G. During 25G run, blackout did not occur but subject was not able to hold air in his lungs despite straining. Thus respiration is the limiting factor at 25G. No pain was experienced. Blurring of vision was reported at higher G levels. Subject was able to operate wrist control stick without impairment of control motion. No disorientation occurred during runs, but first movement after run produced disorientation similar to that of Coriolis effect.

54. CREER, B. Y., STEWART, J. D., DOUVILLIER, J. G.
Influence of sustained accelerations on certain pilot performance capabilities.
Aerospace Med., *33*:1086, 1962.

The authors investigated the ability of trained subjects to tolerate sustained accelerations and to perform a complex tracking task on the Johnsville centrifuge in terms of peak G, position, duration, and rate of onset. A marked deterioration in tracking ability occurred during and immediately after onset of acceleration, most marked in the $-G_x$ and $+G_x$ runs. Deterioration was apparently due, for the most part, to vertigo caused by angular rotation of the gondola. Following onset of acceleration, tracking performance improved rapidly, stabilizing at a constant level. Results showed tentatively that with given vehicle dynamics, a pilot could adequately control an aircraft in a $+G_x$ acceleration field of 14G. At 10G level, there is a small reduction in tracking performance in the $-G_x$ field as compared with the $+G_x$. A marked deterioration is observed above 7G in the $+G_x$ direction. At 7G level, the subjects' ability to see was greatly reduced, and at 8 and 9G level the subjects were on the verge of unconsciousness.

Pilot-performance boundaries in terms of G-time were established, defining the longest period of time a preconditioned pilot, suitably restrained, would voluntarily endure a given G level and perform a control task. A pilot can perform longer in the $+G_x$ or $-G_x$ direction than in the $+G_z$ direction, and again in the $+G_x$ better than in the $-G_x$ position. The respiratory efficiency is greater in the $+G_x$ field than the $-G_x$, but visual problems in terms of watering of eyes were greater in the $-G_x$ field. A pilot could not control a vehicle manually for any extended period at acceleration levels greater than 7 or $8G_z$. And although he could perform at $+14G_x$ level, his performance was poorer than in the $1G_z$ situation.

Performance boundaries as related to vehicle reentry are illustrated, and indicate that a properly positioned pilot could perform at and tolerate acceleration levels expected during reentry with initial entry angle of −8.1°. Tolerance would be doubtful at −8.8°. Rates of onset of 0.1, 0.25, 0.75, and 2G per second were investigated. These demonstrated a fairly rapid decline of pilot's ability to track for acceleration onset rates greater than 0.75G per second. For a vehicle with L/D of 0.5, the maximum acceleration onset rate encountered during 10G is approximately 0.5G per second. How much these data are influenced by pilot vertigo is unknown. Note: Pilots were restrained in the Ames Restraint System.

55. DAVIDSON, S., ROSE, B., STEWART, W. K.
A review of the practicality of, and necessity for, anti-G devices in the RAF with particular reference to the Franks Flying Suit, Mark III.
FPRC 584, 1954.

56. VON DIRINGSHOFEN, H.
Die Wirkung von gradlinigen Beschleunigungen und von Zentrifugalkraften auf den Menschen: Experimentelle Untersuchungen über den Einfluss höher Beschleunigungen auf Blutdruck, Herzchlag und Atmung des Menschen im Motorflug.
Ztschr. F. Biol., *95*:551–566, 1934.

57. DIXON, F., PATTERSON, J. L., JR.
Determination of accelerative forces acting on man in flight and in the human centrifuge.
In Gravitational Stress in Aerospace Medicine, Little, Brown, and Company, 1961.

58. DUANE, T. D.
Observations on the fundus oculi during blackout.
A.M.A. Arch. Ophthal. 51:343, 1954.

An analysis of the term "blackout" is presented. A review of the literature with reference to the two prevalent theories regarding this phenomenon is presented, and it is noted that direct evidence to support either theory was lacking.

In order to provide this needed evidence, an experiment was designed in which the subject experienced positive G, while the observer was exposed to transverse G. Thus, an observer was able to study the fundus oculi ophthalmoscopically while subjects were undergoing blackout. The following direct correlation was established between subjective and objective findings:

Stage	*Subjective*	*Objective*
I	Loss of peripheral lights	Arteriolar pulsation
II	Blackout	Arteriolar exsanguination and collapse
III	Return of central and peripheral lights	Return of arteriolar pulsation and temporary venous distention.

From these findings it was concluded that during positive acceleration blackout is accompanied by a retinal arteriolar ischemia, suggesting that anoxia of the inner retinal cells is responsible for the phenomenon.

The arterial pressure in a human was measured during blackout. It was concluded that there was a correlation between the effective systolic pressure and the symptoms and signs of blackout.

The arterial pressure was measured in a chimpanzee while its fundus oculi was examined during positive G. The ape, likewise, demonstrated the objective signs of blackout at a low G level.

A monkey exposed to positive G showed objective signs of blackout at levels slightly higher than humans. It was concluded that these signs can be used as an index of the threshold of tolerance to positive G in previously unexplored animals.

Intracranial and intraocular pressures of rabbits were recorded during exposures to positive and negative G. There was a decrease in the intracranial and intraocular pressures during positive G, and an increase in these pressures during negative G. However, the changes were of much greater magnitude in the intracranial spaces.

59. DUANE, T. D., BECKMAN, E. L., ZIEGLER, J. E., HUNTER, H. N.
Some observations on human tolerance to accelerative stress. III. Human studies of 15 transverse G.
NADC–MA–5305, 1953. (Authors' Summary)

Five subjects were exposed to 15 transverse G for 5 seconds in the supine position. Also, five subjects were placed in the prone position and exposed to the same accelerative conditions. Blackout and unconsciousness did not occur and the physiological effects produced were of transient nature. An adequately stressed seat was sufficient protection for the supine position. Since the conventional shoulder harness and lap belt were not suitable for levels above 7G when the stress was applied in the prone position, additional thorax and leg barriers were employed.

60. DUANE, T. D., LEWIS, D. H., WEEKS, S. D., TOOLE, J. F.
The effects of applied ocular pressure and of positive acceleration on photic driving in man.
NADC–MA–6214, 1962.

Subjects who demonstrated the phenomenon of photic driving of the EEG were employed in experiments with the ophthalmodynamometer and with positive acceleration on the human centrifuge. When dynamometric pressure was just sufficient to cause pulsation of the retinal vessels, there was a reduction of visual field to approximately 15° in all meridians. Retinal vessels were noted to collapse during diastole. This reduction caused the subjective sensation of grayout, which consisted of loss of all peripheral vision with continued central vision. If a stroboscopic light stimulated the eye while pressure was being applied, loss of photic drive occurred at grayout. On the centrifuge, concurrent with grayout, the arteriolar tree was seen to pulsate, and at blackout complete exsanguination was observed ophthalmoscopically. At the level of grayout, photic driving disappeared, even though subjects continued to perceive stroboscopic light. The occurrence of similar results with pressure and with acceleration eliminates the occipital lobe as the site involved in block of photic driving. The parallel between subjective visual field reduction and observed arteriolar changes both on the centrifuge and with pressure suggests that the same mechanisms operate in both cases.

The authors suggest that these results may indicate that driving is primarily a rod function in the retina. The change in the EEG wave form, observed during the transition from blackout to unconsciousness, could be used as an objective sign indicating the development of unconsciousness during acceleration. During unconsciousness due to positive acceleration, diffuse theta and delta rhythms appear in the EEG. The authors conclude that blackout is primarily a retinal phenomenon and that the block is somewhere between the rods and cones and the visual cortex.

61. EDELBERG, R.
Blood pressures during simultaneous tumbling and deceleration.
Federation Proc., 14:41, 1955. (Author's Summary)

Immediately after emergency ejection, a pilot may be exposed to drag forces as high as 25G in a negative attitude. It has been suggested that tumbling, if rapid enough, may decrease rather than increase the severity of the resulting negative-G hydrostatic effects. The situation was simulated by rotating dogs up to 200 rpm on a spin-table located at the periphery of the human centrifuge. The centrifuge simultaneously imposed "drag" forces up to 10G which changed direction sinusoidally. It was found that superimposed tumbling between 60 and 200 rpm greatly reduced the edema and hemorrhage normally produced by negative G alone. Similarly the vascular damage and engorgement of combined accelerations was less severe than after tumbling alone. Up to ± 10G of drag force, the effects of jostling on the viscera were not significant.

Blood pressures, measured with Gauer-Wetterer intravascular manometers to eliminate the need for hydrostatic correction, were often considerably lower than theoretical pressures. Furthermore, this difference became greater at higher rates of rotation. This effect may, under various circumstances, be due to the low natural frequency of the vascular column, to partial collapse of the column, or to the development of subambient pressures at the center of rotation. The reduction of hydrostatic damage is not due to lower pressures but to the alteration in direction of force with the consequent change in duration of exposure. Circulation is less impaired than in simple tumbling, as evidenced by the smaller reduction in arterial-venous pressure difference.

62. EDELBERG, R.
Hydrostatic effects of combined tumbling and deceleration.
Shock and Vibration Bull., 22:20, 1955.

63. EDELBERG, R.
The physiology of combined accelerations.
In O. H. Gauer and G. D. Zuidema (eds.), *Gravitational Stress in Aerospace Medicine*, Little, Brown and Co., Boston, 1961.

64. EDELBERG, R., WEISS, H. S., CHARLAND, P. V., ROSENBAUM, J. I.
The physiology of simple tumbling.
WADC–TR–53–139, Part I, Jan. 1954.

The tumbling that follows emergency escape from an aircraft by seat ejection or that occurs during prolonged free-fall poses a threat to the escaping crewman. Tumbling was simulated in the laboratory on a horizontal spin-table using anesthetized dogs as subjects preliminary to human experimentation. The axis of rotation was through the heart or at various locations up to 20 cm caudad. The centrifugal forces proved effective in producing peripheral pooling with a consequent reduction in heart filling and cardiac output, as evidenced by the reduced pulse pressure and arterial-venous pressure differences. The decrease in perfusion pressure and the accompanying apnea was enough to produce hypoxia at speeds greater than 140 rpm, as evidenced by oral cyanosis. Concurrently, the elevated hydrostatic pressures were sufficient to produce hemorrhage in the extremities. A tachycardia or bradycardia may occur, depending on the location of the center of rotation. In general, pathology is less when the center of rotation is at the heart than when located at the more caudad positions, but circulation is less impaired as the center is moved caudad.

65. EIBAND, A. M.
Human tolerance to rapidly applied accelerations. A summary of literature.
NASA MEMO 5–19–59E, 1959.

This is a good summary of literature on tolerance to impact analyzed in terms of peak G, plateau G, rate of onset, and duration, according to direction of applied force. It contains useful data, figures, and graphs.

66. ERNSTING, J.
Some effects of oxygen-breathing on man.
Proc. Roy. Soc. Med., 53:96, 1960.

The author describes a clinical syndrome occurring in fighter-aircraft pilots exposed to accelerations of the order of 4 to $5G_z$ while wearing anti-G suits and breathing oxygen. The syndrome is characterized by an attack of coughing and quite frequently by dyspnea, with occasionally deep and ill-localized chest pain, which occurs as the pilot climbs out of his aircraft. The attack may last a few moments or repeated attacks may occur from 10 to 15 minutes after a flight. Incapacitation may occur. Clinical examination may reveal moist sounds over the bases of the lungs and X-ray may show patchy areas of increased density in the lower lung fields, consistent with scattered lobular collapse or areas of edema or infarction. These signs normally clear 18 to 24 hours after completion of the flight. The author advances two hypotheses to explain the syndrome.

(a) During application of G_z in flight, collapse of some basal airways occurs, either from acceleration per se, from descent of the diaphragm and elongation of the lower parts of the lower lobes, or from compression of the lower part of the chest by the anti-G suit. After obstruction of airways, absorption of gas will take place with reduction in volume of closed spaces. When the gas is oxygen, absorption occurs 63 times as fast as when the gas is air. Such collapse is not generally associated with symptoms. Symptoms are probably associated with the reaeration of the collapsed region.

(b) Capillary membrane may be damaged from high alveolar oxygen tension and become more permeable to plasma proteins. The increase in pulmo-

nary capillary pressure in the lower lung occurring with acceleration will then lead to pulmonary edema or perhaps infarction.

Although both mechanisms may play a part, the fact that impairment of the diffusion capacity has been demonstrated in other studies with high alveolar oxygen tension tends to support the second hypothesis.

67. FENICHEL, R. L., KYDD, G. H.
A study of the effects of positive acceleration upon erythrocyte hydration in human subjects.
NADC–MA–5904, 1959.

Human subjects were employed in a study of the effects of positive acceleration upon erythrocyte hydration. Venous blood samples were obtained just before the acceleration series was begun, after the third centrifuge run ($2.5G_z$), and immediately after the subject was exposed to the last acceleration run in the G series ($5.5G_z$). The acceleration exposure began at the 1.5G level and increased in ½G increments, with a 5-minute rest period between runs. The exposure was terminated when the subject lost peripheral vision. The unprotected subjects used in this study reached 5.5G, on the average, before losing peripheral vision.

A decrease in mean corpuscular hemoglobin concentration (MCHC) was observed. A trend toward an increase in mean corpuscular volume was noted. The fall in MCHC indicated that after G exposure the erythrocytes of human subjects were increased in size. Fluid had shifted into the red blood cells. Comparison of the results obtained with humans at relatively low G levels with the results obtained from monkeys at overlapping and higher G levels indicated that in both species the MCHC decreased.

68. First U.S. Manned Orbital Space Flight, February 20, 1962.
Manned Spacecraft Center, NASA.

Both maximum exit acceleration and maximum entry acceleration were 7.7G. Pulse rate from lift-off to spacecraft separation reached a maximum of 114 per min. During reentry, the highest rate was 134 per min and the mean rate 109 per min. This rate was the highest noted during the mission. Rates were consistently higher in flight than in centrifuge simulation. The astronaut commented that linear acceleration buildup is pleasanter than radial acceleration, as in the centrifuge. He also pointed out that acceleration was not a major problem and that he could communicate well up to the maximum of 7.7G at insertion.

69. FRANKENHAEUSER, M.
Effects of prolonged gravitational stress on performance.
Acta Psychologica, 14:92, 1958.

The author exposed approximately six subjects in each series to $3G_z$ for 2 to 10 minutes at a rate of onset of approximately 1G per 3 seconds. Studies included eight different psychological tests:

(1) Visual choice reaction time, a complex light canceling test. A small but significant number of errors occurred under all experimental conditions.

(2) Visual acuity, using a C-type chart. A decrement in performance occurred corresponding to loss of 16% acuity.

(3) Accuracy of movement, a continuous pursuit task. Performance decreased significantly during exposure.

(4) Perceptual speed, identification of a test figure from a group. No errors were obtained.

(5) Stroop test, an estimate of reading speed, color naming, and reaction to self-induced stress. Showed significant decrement in reading speed and color naming.

(6) Subtraction test, serial from 100. Time taken increased significantly during exposure to 3G.

(7) Multiplication. Ability was impaired during exposure.

(8) Time perception, estimating duration of half-duration. Speedup of function was more pronounced during influence of 3G than under normal conditions.

Previous experience did not appear to influence differences in performance. However, effects on performance in the same tests appear to be subject to adaptation, regardless of the number of previous runs.

70. FRANKS, W. R.
Acceleration or "G."
In An Aeromedical Handbook for Aircrew, RCAF Pamphlet 69, 1960.

71. FRANKS, W. R., KERR, W. K., ROSE, B.
Some effects of centrifugal force on the cardiovascular system in man.
J. Physiol., 104:9P–10P, 1945. (Authors' Summary)

By means of the centrifuge, the effects of increased positive G on the cardiovascular system were studied in 72 subjects during 690 tests at 2 to 10G. Kodachrome motion pictures showed, as increased G was applied, blanching of the face and distension of the superficial leg veins, which persisted until a few seconds after the G began to diminish. The leg veins then reverted to normal, but the facial blanching was followed by flushing which lasted 10 to 20 seconds.

The ear opacity began to decrease with the onset of increased G, reaching a minimum 4 to 6 seconds after G became constant. At 0.5 to 3 seconds after the G began to decrease, the ear opacity rapidly increased. The increase continued above the initial level, coincident with the facial flushing. The decrease in ear opacity was directly but not quantitatively related to the amount of G applied.

The heart rate increased rapidly with the onset of increased G, attaining a maximum of 120 to 190 beats/min, depending upon the amount of G and its duration. When the maximum G was maintained

more than 10 to 20 seconds the maximum heart rate was relatively constant until the G was reduced. With the reduction of G in short runs there was a delay of 2 to 5 seconds before the heart rate suddenly fell to below its initial resting level. This bradycardia coincided with the flushing and increased ear opacity, and was frequently followed by secondary rise in rate.

Electrocardiograms from chest electrodes over base and apex of the heart showed the following changes during increased G. The PR interval was shortened. The overall amplitude of the QRS complex decreased, usually with the main deflection downward. The T wave flattened and sometimes disappeared. As the G was reduced, the PR interval and QRS complex reverted to their original form, but the T wave became greatly increased in amplitude and sometimes biphasic for 2 to 5 minutes. During this period sinus arrhythmia and, more rarely, ventricular extrasystoles appeared.

Anterior-posterior X-ray films of the chest (1 sec exposure) taken during increased G showed a marked reduction in cardiac shadow as compared to that of control films.

The circulatory changes described in this paper could not be related to the level of G at which a subject would black out or lose consciousness. However, the pooling of the blood in the lower extremities, reduction in cardiac shadow, facial blanching, decrease in ear opacity, and associated changes in heart rate appear to be dependent variables and throw some light on the action of increased positive G on man.

72. FRANKS, W. R., KERR, W. K., ROSE, B.
Some neurological signs and symptoms produced by centrifugal force in man.
J. Physiol., 104:10P–11P, 1945. (Authors' Summary)

The neurological effects of centrifugal force in man were studied in 542 subjects during 5,544 test runs at $+2G_z$ to $+10G_z$ in the centrifuge. As a measure of performance during exposure to centrifugal force, the reaction time for manual responses to visual and auditory stimuli was recorded for 7,853 stimuli during 626 tests at 2 to 8G on 35 subjects, but it was not significantly increased, except for visual stimuli immediately before blackout.

As a result of exposure to increased G, however, convulsions frequently occurred, usually after loss of consciousness. (52% of 230 subjects had convulsions in 40% of 591 tests producing unconsciousness.) They were usually slight, clonic seizures involving all or some of the extremities, face, and trunk. Less commonly, severe generalized convulsions were observed. These varied greatly and sometimes included a brief tonic state with neck and trunk in extension, occasionally with arms extended in pronation and legs drawn up in flexion. Conjugate movements of head and eyes to one side were sometimes observed. Usually violent jerks of the extremities and trunk terminated the seizure in 2 to 5 seconds. Finally, a small number of slight convulsions were noted in fully conscious subjects. Dreams were frequently experienced, usually in association with convulsions. Paresthesias, confused states, amnesia, and, more rarely, gustatory sensations were noted with blackout and loss of consciousness, either with or without convulsions. Incontinence was never observed.

73. FRAZER, J. W., REEVES, E., HARDY, J. D., SHEPLER, H. G.
Adaptation to positive acceleration.
NADC–MA–5818, 1958.

To investigate the occurrence of adaptation, the authors exposed rats to 19.2G in two groups. One group had been conditioned to 2G for 5 days a week for 6 weeks, and one to 12G 5 days a week for 6 weeks. The time of survival was determined by ECG pulse rate. The 2G group was significantly lower and the 12G higher in survival time than control animals similarly stressed apart from acceleration. Mean survival time at 20G for cage-stressed controls was 1,281 seconds; for rats preconditioned at 2G it was 969 seconds, and for rats preconditioned to 12G it was 2,011 seconds. Time to peak G in all conditioning accelerations was 12 seconds.

74. FRYER, D. I.
Physiologic effects of exposure to ram pressure.
Aerospace Med., 33:34, 1962.

Using an underwater centrifuge, the author studied the effects on man of dynamic exposure to ram pressure. Breath holding could safely be maintained in spite of measured rise of pressure in the thorax to as much as 5.25 psi. The author derived a series of values for the forces acting on the arms and legs up to the equivalent of 515 and 416 knots indicated airspeed. Petechiae and bruising were produced during the runs; the author noticed the similarity to "squeeze" found in diving, and related the results to those of Stapp in his sled experiment.

75. GAMBLE, J. L., SHAW, R. S., HENRY, J. P., GAUER, O. H.
Cerebral dysfunction during negative acceleration.
J. Appl. Physiol., 2:133, 1949.

Six humans were studied in the upright seated posture during exposure to accelerations up to $-3G_z$. ECG's were taken and arterial and venous blood pressures recorded at head level. Dogs and rabbits were exposed to accelerations up to $-7G_z$. Exposures were of 15-second duration for humans and of 2-minute duration for animals. Human venous pressure changes were measured at the frontal vein, and arterial changes in the radial artery held at the forehead. Pressures in dogs were recorded directly from the carotid artery and jugular vein. ECG records (modified L 11) showed arrythmias of various types compatible with marked vagal stimulation, beginning with the onset of acceleration and gradually returning to normal with the end of acceleration.

Blood pressure recordings indicated a rise in arterial and venous pressure of about 70 to 90 mm. Although initially showing a rapid parallel rise, as the run progresses there is a diminution of arterial pressure and steady rise in venous pressure, perhaps from continued drainage of blood from legs. Thus, the A–V pressure differential across the brain decreases with increasing time of application.

In animal subjects the most dramatic effect was pronounced bradycardia, beginning 3 seconds after onset of acceleration. Periods of asystole as long as 10 to 20 seconds and heart block of varying degrees with nodal and ventricular escape were noted. Bigeminal extrasystoles and idioventricular rhythm were also recorded. Such changes could be abolished following section of the vagus nerve. Arterial pressure at the level of the carotid sinus rose from about 100 mm Hg to about 260 mm, while that in the jugular vein rose to about 100 mm.

EEG's taken at $-7G_z$ for 1 minute showed a delta rhythm indicative of brain injury.

The bradycardia, long periods of asystoles, and heart block with arrythmia are considered by the authors to result from vagal stimulation by way of the carotid sinus and may be so severe that circulation to the brain becomes inadequate to maintain consciousness.

76. GARROW, G. S.
Positive acceleration and the release of antidiuretic hormone in man.
FPRC 1129, 1960.

White et al.[182] (1926) showed that assumption of the erect posture after recumbency reduces the rate of urine secretion. Silvette and Britton (1948) found a similar effect on exposure of rats to acceleration, as did Stauffer and Errobo-Knudsen[162] (1953) in man. Taylor and Noble demonstrated the appearance of an antidiuretic substance in the urine of man in stress situations (1950), but failed to find it in those who had blacked out on the centrifuge (1953). The author maintained three experienced subjects with water load of 250 ml for 3 hours. At the middle of this period they were exposed for 10 minutes to 2G positive acceleration. One subject always had antidiuresis (11 times out of 11), one subject did not (0 time out of 2), and one sometimes did (1 time out of 2). Water retention was accompanied by release of antidiuretic hormone, marked but transient retention of sodium, and less marked but equally transient retention of potassium and total solids.

The author suggests that antidiuretic effect may be blocked by release of adrenalin, but did not measure adrenalin secretion. He notes that the response is found only under positive acceleration. He considers other factors that might be involved in the antidiuresis, namely, local reduction in perfusing pressure, renal vasoconstriction, reduction in cardiac output, and factors tending to decrease filtration pressure, but dismisses them with justifiable argument.

77. GAUER, O. H.
Die Atemmechanik unter Beschleunigung.
Luftfahrtmedizin, 2:291–294, 1938.

78. GAUER, O. H.
The physiological effects of prolonged acceleration.
In German Aviation Medicine, World War II, Vol. I. Superintendent of Documents, U. S. Government Printing Office, 1950.

79. GAUER, O. H.
In O. H. Gauer and G. D. Zuidema (eds.), *Gravitational Stress in Aerospace Medicine*, Little, Brown and Co., Boston, 1961.

A collection of papers on the physiological effects of accelerations.

80. GAUER, O. H., HENRY, J. P., SIEKER, H. O.
Cardiac receptors and volume control.
Progress in Cardiovascular Diseases, 4:1, 1961.

As part of a general paper considering cardiac receptors and fluid volume control, the authors discuss the evidence for localizing the cardiac receptors. In one series of experiments, pulmonary artery pressure was increased by approximately 15 cm of water by infusing an aqueous suspension of plastic beads into the superior vena cava. In another, a wire loop was introduced around the pulmonary veins on both sides. By controlled tightening, the desired degree of pulmonary vascular hypertension, from 22 to 41 cm of water, was induced for 20 to 30 minutes at a time. Both these experiments caused obstruction to blood flow with some decrease in cardiac output, and caused a slight decrease in urine flow in 14 experiments with 10 dogs. In a further study, a small rubber balloon was introduced into the left atrium of dogs through the atrial appendage. When distended by water, a controlled rise in left atrial and pulmonary intravascular pressure of 14 to 20 cm of water was obtained. In 37 such distentions in 13 dogs, an increase in urine flow was obtained in all but 4 cases.

These experiments showed that engorgement of the pulmonary vascular bed was effective only if the left atrium was included in the congested area. Thus, the highly distensible great veins and atria appeared to be the most appropriate locations for volume receptors, while the left atrium in particular is one sensitive area concerned with antidiuretic hormone release.

81. GAUER, O. H., RUFF, S.
Die Erträglichkeitsgrenzen für Fliehkräfte in Richtung Rucken-Brust.
Luftfahrtmedizin, 3:225–230, 1939.

82. GELL, C. F.
Table of equivalents for acceleration terminology.
Aerospace Med., 32:1109–1111, 1961.

The table described is recommended for general international use by the Acceleration Committee of the Aerospace Medical Panel, AGARD.

83. GELL, C. F., HUNTER, H. N.
Physiological investigation of increasing resistance to blackout by progressive backward tilting to the supine position.
NADC–MA–5406, 1954.

The purpose of this project was to demonstrate that when fully supine, exposure to 15 transverse G can be tolerated for 5 seconds with no indication of impending blackout. It further demonstrates that with a 77° backward tilt the antiblackout protection did not exceed that afforded by an inflated anti-G suit with the subject in the upright seated position.

A close relationship was observed between the degree of backward tilt of the seat, the vertical angle of the retinal-aortic dimension, and the degree of blackout protection afforded.

84. GLAISTER, D. H.
Breathing.
Nature (London), *192*:106–108. 1961.

A review is given of two papers presented at a symposium on "breathing" held at the meeting of Section I of the British Association for the Advancement of Science on Sept. 4, 1961. The first paper studies the problems of air temperature and pressure at 40,000 feet, and includes effects of explosive decompression from 8,000 to 38,000 feet. A design for oxygen masks to allow proper breathing under decompression is described. The second paper is concerned with the effects of posture on breathing under positive acceleration. The mechanical effects on the lungs, viscera, and diaphragm under accelerations of 3G are described, and it is shown that the total efficiency of the respiratory process decreases during positive acceleration. This would probably result in a marked limitation in tolerance to exercise under continuous positive acceleration.

85. GLAISTER, D. H.
Pulmonary gas exchange during positive acceleration.
FPRC–1212, Apr. 1963.

Pulmonary gas exchange and lung function have been studied in man subjected to positive (tailward, $+G_z$) acceleration in the sitting position.

During runs at 2 to 3G lasting ½ to 5 minutes, the oxygen uptake fails to rise, or may even fall, despite the fact that an oxygen debt is being built up which is only repaid on return to 1G. The oxygen debt is increased by increasing either the duration or the severity of acceleration, and is slightly decreased when an anti-G suit is worn. Breathing 100% oxygen has no effect on the development of the debt, which may reach 500 to 600 ml without an increase in oxygen uptake during the period of acceleration. Moderately severe exercise has to be performed during acceleration before the oxygen uptake is raised at 2 to 3G, and following such a run the oxygen debt may reach 740 ml.

Marked inequalities in ventilation and perfusion are inferred from the pattern of carbon dioxide excretion during tidal and prolonged expirations at 3G, and the effect of these on the reserves of oxygen and carbon dioxide in blood and tissues is discussed. A falling oxygen reserve secondary to these changes in lung function could explain the decrease in oxygen uptake during acceleration, but could be responsible for only a small fraction of the oxygen debt.

The bulk of the oxygen debt is probably due to the building up of products of anaerobic metabolism, but the reason for anaerobic metabolism at these low levels of acceleration is unclear. A number of other theories to explain the development of an oxygen debt are also discussed, but none provides a satisfactory explanation.

86. GOODALL, M., BERMAN, M. L.
Urinary output of adrenaline, noradrenaline, and 3-methoxy-4-hydroxymandelic acid following centrifugation and anticipation of centrifugation.
J. Clin. Invest., *39*:1533, 1960.
(Abstract from STL–9990–6302–KU–000)

Nine normal males were centrifuged at a rate of 1G per 5 seconds to 12G, or were given a mock centrifuge ride at 2 rpm (equivalent to 0.02G forward acceleration) for 3 minutes, respectively. Each subject was unaware whether he would receive a real ride or a mock ride, and it was therefore possible to measure the sympatho-adrenal response to both centrifugation and anticipation of centrifugation. Under high gravitational stress, increased urinary adrenaline release seems to be largely related to the emotions, while noradrenaline release seems more closely related to the physical changes (hemodynamics) produced by centrifugation. Following the increased release of adrenaline and/or nonadrenaline, there is a commensurate rise in the urinary output of their common metabolic product, 3-methoxy-4-hydroxymandelic acid.

87. GRAY, R. F., WEBB, M. G.
High G protection.
Aerospace Med., *32*:425, 1961.

Material for this paper is taken from an earlier paper by the same authors (Gray and Webb,[88] 1960). It includes a discussion of the theory of water-immersion protection and references the early German work and the development of the Franks flying suit. It also describes two studies, one with the Mayo tank and the other with a rigid, semiform-fitting G-capsule (the Iron Maiden). In the former study a subject in the seated position, while submerged and holding the breath, could tolerate $+16G_z$ without dimming of peripheral vision. With no water in the tank, peripheral vision was lost at $+3.25G_z$. Tolerance was limited by air being forced from the chest.

In the second study, submerged in the Iron Maiden and breathing air at a pressure just above the expected hydrostatic head, subjects could tolerate a peak of $-25G_x$ applied in the pattern of a 25-second haversine. Further pressurization was limited by

ear and frontal sinus pain. With breath holding, one subject attained $-26G_x$, a second subject $-28G_x$, and a third subject $-31G_x$ with minor disablement, namely sinus and abdominal pain and flecks of nasal hemorrhage. No impairment of vision was observed and no eye pain. Postrun clinical examinations indicated no congestion within the chest, and no ECG abnormalities were observed. Free movement of limbs was possible even under very high acceleration.

88. GRAY, R. F., WEBB, M. G.
High G protection.
NADC–MA–5910, 1960.

In this paper the authors discuss problems of tolerance to sustained acceleration above the 5G level. By a theoretical analysis, confirmed by practical experimentation with analog models, they define the protection given by water immersion systems. They describe experiments using the Mayo tank and a G-capsule in which seated human subjects were exposed to high acceleration loads with application in the $-G_x$ vector while submerged. (See Gray and Webb,[57] 1961.) They also list and comment on other methods of protection against sustained acceleration: Partial supination, straining with chest and abdomen expanded, breath holding, G-suit inflation, custom molded couches, water-filled exposure suit plus G suit plus molded couch, taping of buttocks and triceps, and elevation of legs.

89. HALLENBECK, G. A.
Effects on man of repetitive exposure to centrifugal force.
Federation Proc., *4*:29–30, 1945. (Author's Summary)

This study was undertaken to determine the response of subjects in increased G when exposures follow one another at time intervals brief enough that the response to one exposure could be conditioned by previous trials. The Air Technical Service Command centrifuge was operated such that six 10-second episodes of 4.2G, a level sufficient to produce marked visual symptoms in the chosen subjects in a single test run, were delivered in sequence. Time intervals from the end of maximal G in one episode to the beginning of maximal G in the next were set at 4.7, 9.6, 19.4, and 29.1 seconds. Continuous exposure for 60 seconds was also imposed. During continuous 60-second exposures, six subjects who suffered either loss of peripheral vision or blackout during the first 10 or 15 seconds showed varying degrees of improvement in vision in the second and subsequent episodes of each series. This improvement was constant and marked when the interval between exposures was 10 seconds or less, and less consistent when the interval was 15 or 30 seconds.

90. HAM, G. C.
Effects of centrifugal acceleration on living organisms.
War Med., *3*:30, 1943.

A very fine review article detailing the work in acceleration prior to 1943, with particular reference to the early German work. Should be consulted for references to historical information. Deals with tolerance and physiology under positive, negative, and transverse acceleration for animals and man in aircraft centrifuges and tilt tables. Covers cardiovascular studies, respiratory studies, X-ray studies, blackout, mental efficiency and performance, prophylaxis, and protection.

91. HEADLEY, R. M.
Human tolerance and limitations to acceleration.
In P. Bergeret (ed.), *Bio-Assay Techniques for Human Centrifuges and Physiological Effects of Acceleration*, Pergamon Press, New York, 1961.
AGARDograph No. 48.

This paper describes tolerance and limitations as demonstrated at the Aerospace Medicine Laboratory, Wright-Patterson Air Force Base, in terms of headward, forward, lateral, footward, backward, and complex acceleration.

Lateral: Five subjects were exposed to left and right lateral acceleration to a maximum of 7G in both vectors. Exposures were terminated because of severe vascular engorgement with pain in dependent forearm and elbow. Chest pain and tugging were occasionally present. X-ray at 6G in left lateral acceleration showed marked displacement of heart to right and some clockwise rotation of heart, radioopacity of right lung, left hemidiaphragm displaced and flattened, right hemidiaphragm probably normal, left lung blanched. In right sideways acceleration changes were identical but opposite in direction. Along with displacement of barium-filled stomach against lateral abdominal wall, there was movement of liver to left.

Complex acceleration: Human subjects (number not stated) were exposed to backward acceleration of 8G with pitch of 1 cps through half-amplitude of 20°. No significant evidence of injury was demonstrable. Five primates were exposed to simulated deceleration in forward-facing position up to 20G with sine-wave pitch oscillations up to 60 seconds through half-amplitude of 20° at 3 and 5 cps. Maximum resultant accelerations, backward 40G, footward 10G, headward 15G, were recorded from skull. After exposure all animals were disorientated but could make coordinated movements. No irreversible injuries were demonstrated live or postmortem.

92. HENRY, J. P., GAUER, O. H., KETY, S. S., KRAMER, K.
Factors maintaining a cerebral circulation during gravitational stress.
J. Clin. Invest., *30*:292, 1951.

The authors observe that consciousness is usually lost when mean cerebral blood pressure falls to 25

mm Hg, but during acceleration sufficient to produce blackout, alertness may be maintained in spite of such mean pressures. They measured arterial and cerebral venous oxygen saturation and arterial pressure at head level, as well as cerebral venous pressures, in three subjects under positive acceleration ranging from 1 to 4½G for 1 to 2 minutes. Venous saturation remained almost unchanged in spite of large falls in cerebral arterial pressure, suggesting that some other factor was maintaining cerebral blood flow. Pressures ranging from 20 to 60 mm Hg below ambient were found in the jugular bulb. This could maintain consciousness by sustaining an A–V pressure differential, or by producing a combination of such an effect with passive cerebral vasodilatation. The authors also suggested that active cerebral vasodilatation may occur during prolonged exposure to gravitational stress in the erect posture.

93. HENRY, J. P., GAUER, O. H., REEVES, J. L.
Evidence of the atrial location of receptors influencing urine flow.
Circul. Res., *4*:85, 1956. (Authors' Summary)

Receptors believed responsible for the diuresis of negative pressure breathing have been located by observing the effect on urine flow of a stepwise engorgement of the intrathoracic vascular bed. Distension of the pulmonary arterial tree (injection of plastic beads) and of the entire pulmonary circulation (snaring of the pulmonary veins) were without effect. A diuresis was, however, elicited by expansion of a balloon in the left atrium. It is concluded that stretch receptors in the left atrium and terminal pulmonary veins are instrumental in a mechanism linking changes in the actively circulating blood volume with homeostatic responses of the kidney.

94. HERSHGOLD, E. J.
Roentgenographic study of human subjects during transverse accelerations.
Aerospace Med., *31*:213, 1960.
(Abstract from STL–9990–6302–KU–000)

Chest and abdominal roentgenograms of human subjects undergoing forward acceleration at 6 and 12G, and left and right sideward accelerations at 6G, demonstrate the sensitivity of the pulmonary circulation to simulated increased gravity and the vulnerability of the mediastinal and abdominal organs to displacement in this state.

95. HERSHGOLD, E. J., STEINER, S. H.
Cardiovascular changes during acceleration stress in dogs.
J. Appl. Physiol., *15*:1065, 1960.

Dogs were exposed to levels of 3 and 4G ($+G_z$) at a rate of onset of 1G per 15 seconds and to 4 and 6G ($+G_x$) at a rate of onset of 1G per 5 seconds. Cardiac output, blood pressure, and heart rate were measured, and stroke volume and peripheral resistance were calculated. Measurements were determined 1 minute after onset of acceleration. Results showed cardiac output reduced in headward acceleration, but stable or increased in transverse acceleration. Mean arterial blood pressure was slightly reduced in both vectors, more so in the positive. Heart rate was increased in both vectors. Stroke volume in the positive vector was reduced 65%. In transverse acceleration there were insignificant changes in stroke volume. Peripheral resistance increased 70% in the positive vector but decreased 26% in the transverse vector. Reduced cardiac output in the positive vector probably results from expansion of dependent portions of the venous reservoir, but since the heart is displaced downward and lengthened during positive acceleration, this may result in obstruction to the venous entry.

96. HESSBERG, R. R.
Acceleration environments pertinent to aerospace medical research.
In Human Acceleration Studies, NAS–NRC Pub. 913, 1961.

Space flights may be divided into five phases: I, Launch; II, Orbit; III, Reentry; IV, Recovery (high-drag system only); V, Landing. Two basic vehicle configurations exist: high-drag (Mercury Spacecraft) and high-lift (Dyna-Soar).

Phase I: Launch accelerations are largest in magnitude because of denser atmosphere. Onsets are low. Atlas or Titan can produce forces peaking at 16G, being above 10G for 40 seconds, with 20 to 30 seconds to and from 10G. Can be designed to produce 8 to 9G peak, with 60 to 80 seconds above 6G and a total duration of 160 to 180 seconds. A G/time history of 7G for 180 to 200 seconds for Nova, and 5G for 300 to 320 seconds for Saturn can be expected. Onsets for second- and third-stage boosters will be higher. The author notes the offsets should be similar to onsets.

Phase II: Essentially a weightless situation, but the accelerations associated with navigation and attitude adjustment may have a detrimental effect on vestibular function.

Phase III: A blunt body uncontrolled could obtain a peak of 300 to 400G. High-drag systems can be designed to experience 12 to 16G for about 10 seconds at maximum with gradual buildup to peak G over 30 to 50 seconds. High-lift design produces negligible G loads.

Phase IV: G forces during parachute deployment and inflation are relatively low, being 9 to 11G for 2 to 3 seconds.

Phase V: Introduces problems of oblique G, the physiological effect of which is almost uninvestigated.

Escape: In the worst situation, that is, at high velocity during launch and still within the atmosphere, necessary thrust is calculated to produce 22 to 26G at 2,000 to 4,000G per second for 8 to 12 seconds accompanied by oscillation.

97. HOLDEN, G. R., SMITH, J. R., SMEDAL, H. A.
Physiological instrumentation systems for measuring pilot response to stress at high G and zero G.
Abstracted in *Aerospace Med.*, *32*:235, 1961. (Authors' Summary)

An airborne physiological instrument system reported in NASA TN D–351 has been modified and additional tests have been made in the University of Southern California and AMAL centrifuges and in an F–104B airplane. These tests covered various levels of acceleration from zero to 7G. The measurements made were, in part: ECG, blood pressure, pulse wave, respiration rate and volume, and carbon dioxide content of expired air. The data from a three-lead electrocardiograph were recorded, using a unique balanced transistor amplifier. Systolic and diastolic blood pressures were measured by using an automatic sequencing occluding arm cuff and microphone stethoscope. Pulse wave on the wrist was obtained with a vasochromograph and ac amplifier. Several methods were used to measure respiration rate, and respiration volume was measured with a WEDGE spirometer. The expired air was analyzed for carbon dioxide content with a very much modified Bechman LB–1 gas analyzer.

The quantitative effects of short-term periods of zero G on pilot control performance were determined by measuring the tracking accuracy, the equivalent analytical transfer function, and the physiological condition of a subject in the rear seat of an F–104B airplane being flown in a 60 to 80 second zero-G trajectory. A tracking task played back from a tape recorder was presented to the subject on an oscilloscope. The subject used a sidearm controller to attempt to wipe out his tracking error. A small airborne analog computer computed the simulated airplane's response to the control motion and changed the tracking display accordingly. The experiment was repeated and thus affords a direction comparison with a study of pilot control behavior previously conducted on a ground-based simulator and a centrifuge.

98. HOWARD, P.
Changes in the cardiac output during positive radial acceleration.
J. Physiol., *147*:49P, 1959. (Author's Summary)

It is known that positive radial acceleration (centrifugal force acting in the head-to-foot direction) produces profound changes in the dynamics of the circulation. The output of the heart is likely to be affected by these changes but hitherto no experimental confirmation of this assumption has been presented.

The direct Fick method was used to determine the cardiac output in two subjects. A polythene catheter was inserted into the right atrium via an antecubital vein, its position being confirmed with examination of the pressure wave form. The subject lay supine on the end of the human centrifuge, with the legs extended. Expired air (5 min collections into a Douglas bag) and mixed venous blood from the auricle were collected under the following conditions: After 10 minutes at rest, after the centrifuge had been accelerated to a constant speed, and after 15 minutes' rest with the machine stationary. In one case the resultant acceleration used was 2.0G and in the other it was 2.4G. A sample of blood from the femoral artery was obtained immediately after the machine had come to rest. The gas and blood samples were analyzed by the Haldane and Van Slyke methods, respectively, and from the results the oxygen consumption and cardiac output were calculated.

At 2G the output was reduced to approximately 68% of the resting value, and at 2.4G it fell to about 60%. Because of the concomitant increase in heart rate the changes in stroke volume were greater still. In both cases the oxygen consumption rose by about one-third during the exposure.

These results may be compared with those obtained after tipping from the horizontal to the erect posture (equivalent to a radial acceleration of 1G) which produces an average decrease of cardiac output of 25%.

99. HYDE, A. S., CHERNIACK, N. S., LINDBERG, E. F., WHATELY, D.
Cardiorespiratory responses of flying and nonflying personnel to different vectors of acceleration with correlation of these responses to other variables.
BioMed. Lab. AMRL–TDR–62–151, 1962.

A group of astronaut candidates and a group of nonrated centrifuge panel members underwent headward ($+G_z$) and forward ($+G_x$) accelerations on a contoured net seat. No significant difference was found in the responses of the two groups. Two of the test pilots reached $+9G_z$ acceleration without blackout and without G suit. No correlation was found between pulse rate and blackout level. Vital capacity decreased about 50% in both groups at $+5G_z$. Forward acceleration to $+12G_x$ caused little change in pulse rate and a few minor ECG abnormalities, despite respiratory maneuvers. Vital capacity decreased with increasing forward accelerations. Change in back angle from 0° to 12° did not significantly influence rate of decrement in vital capacity. Half-second forced expiratory capacity represented increasing portion of total vital capacity as forward acceleration increased. Blackout tolerance during headward acceleration and respiratory performance during headward and forward acceleration did not correlate with an extensive number of anthropometric measurements and physical fitness tests, nor was correlation found between $+G_x$ and $+G_z$ acceleration responses and measurements made during other stressful situations.

100. ISAKOV, P. K., STASEVICH, R. A.
Speed, Acceleration, Weightlessness.
FTD–MT–63–103, 1964.

101. JASPER, H. H.
Centrifuge experiments with animals.
Montreal, Neurological Institute, McGill University, Montreal, Canada, 1947.

102. JONGBLOED, J., NOYONS, A. K.
The influence of acceleration upon the circulation.
Arch. Ges. Physiol. (Ger.), *233*:67, 1933.

103. KEIGHLEY, G., CLARK, W. G., DRURY, D. R.
Flicker fusion frequency measurements on man subjected to positive acceleration on a human centrifuge.
J. Appl. Physiol., *4*:57–62, 1951.

104. KISELEV, A. A.
Some peculiarities of hemodynamics and gas exchange in pulmonary circulation under transverse ventrodorsally directed accelerations. Experimental studies on animals.
Problemy kosmicheskoy biologii. v. 2, ed. by N. Sisakyan and V. Yazdovskiy, Moscow, Izd-vo AN SSSR, 1962, pp. 231–237.

Changes in the pulmonary circulation of dogs under the effects of ventro-dorsal accelerations of 3, 6, and 9G were investigated. In all, 66 experiments were conducted on dogs of both sexes weighing between 6 and 9 kg. The following indicators were determined: ECG, the pressure in the right ventricle by means of probes, the rate of pulmonary circulation, the oxygen volume concentration in arterial blood, the respiration rate, and the pulse rate. For accelerations higher than 3G the pulse pressure in the right ventricle increases; for 9G this increase becomes regular. It was found that the volume concentration of the oxygen is closely related to the blood-volume rate in the pulmonary circulation; the former varies in direct proportion with the latter. In all the cases a decrease in the blood-volume rate was accompanied by a decrease in the oxygen volume concentration of the arterial blood.

It was concluded that acceleration leads to marked changes in the hemodynamics of the pulmonary circulation, which is a major factor in maintaining an adequate blood oxygenation level in the lungs. One of the most plausible mechanisms for the maintenance of this level is the progressive depositing of blood in the arterial system of the lung, accompanied by unequal systolic volumes of right and left ventricles. Notwithstanding the considerable efficiency of such a mechanism of compensation, the level of oxygenation becomes lower after 1 to 1½ minutes of acceleration, the final level depending on the magnitude and duration of the acceleration.

105. KOTOVSKAYA, A. R., YUGANOV, YE. M.
The effect of prolonged transverse acceleration on animals.
In Problems of Space Biology, Vol. 1. Ed. N. M. Sisakyan, USSR Academy of Sciences Publishing House, Moscow, 1962.
NASA TT F–174.

This paper begins with a brief discussion of previous work in transverse acceleration by American and European investigators. The authors describe an investigation with 14 dogs exposed to $+G_x$ acceleration up to 10G for durations of 3, 6, and 15 minutes. They recorded ECG, respiratory rate from changes in chest perimeter, and maximum blood pressure by an oscillator method during periodic constriction of the carotid artery lifted into a skin flap. They noted indications of apprehension in the animals with start of acceleration. During repeated experiments, apprehensive reactions in the blood pressure and respiration were observed before the effect of acceleration could be felt. During acceleration, the animals showed a marked increase in salivation which continued for some time after acceleration had ceased.

With onset of acceleration, the heart rate increased 1.5 to 2 times, and it remained at that level during acceleration. After acceleration ceased there was a gradual return to the original level within five to ten minutes. Normal sinus arrhythymia either disappeared completely or became less significant during acceleration. Respiration rate increased 1.5 to 2 times with onset of acceleration and continued to increase to 3 times the original value. When acceleration ceased, respiratory rate slowed to the original level within 5 minutes. Blood pressure increased by 50 to 80 mm Hg with onset of acceleration, and persisted at that level through the period of acceleration. With cessation of the acceleration, a marked gradual lowering of arterial pressure occurred over a period of about 5 to 10 minutes. The dog Layka showed an increase in heart rate with onset of acceleration from a control of 140 to 200 beats per minute. The rate increased further to 290 and was maintained within the range of 220 to 290 throughout the period of acceleration. Respiration rate, with onset of acceleration, increased from a control of 20 to 53 per minute, and maintained a rate of 30 to 35 per minute during acceleration. Arterial blood pressure increased with onset of acceleration from a control of 180 mm Hg to 210 to 230 mm Hg and was maintained at this level throughout the acceleration.

106. KOVALENKO, YE. A., POPKOV, V. L., CHERNYAKOV, I. N.
The effect of transverse acceleration on oxygen tension in brain tissue.
Sechenov Physiol. J. USSR, *49*:1145, 1963. (FTD–TT–63–1215).

The authors measured directly, by platinum electrodes in the brain of dogs, the relative Po_2 of brain

tissue under $+G_x$ acceleration as compared with pre-acceleration control. They found an initial increase in P_{O_2} with onset of acceleration which was abolished by anesthesia, and thereafter a progressive fall in P_{O_2} related to magnitude and duration of acceleration, position of dog's head with respect to trunk, and individual variation. Specific data are presented.

107. LAMBERT, E. H.
The physiologic basis of "blackout" as it occurs in aviators.
Federation Proc., *4*:43, 1945.

Blackout in aviators is a temporary loss of vision without disturbance of consciousness, occurring during exposures to high positive acceleration. Experiments were designed to determine the role of the retina in the origin of this phenomenon.

1. At 1G (gravity) temporary loss of vision was produced by application of air pressure to the eyeball, using special masks. The effective systolic arterial pressure to the eye (systolic pressure at head level minus the applied eye pressure) at which symptoms occurred was, in millimeters of mercury: Vision dim, 49 to 30; peripheral vision lost, 32 to 20; vision completely lost, 21 to 0. These visual changes were the same in latent period and progress of development, and occurred at the same level of effective blood pressure, as the visual changes that occur at high positive accelerations on the human centrifuge.

2. On the centrifuge, application of 20 to 30 mm Hg pressure to the eyeball lowered by 1G the threshold acceleration at which visual changes occurred. This pressure corresponds to the fall in systolic arterial pressure per G found to occur at head level during exposure to acceleration.

3. The application of 30 to 40 mm Hg suction to the eyeball prevented the occurrence of blackout during exposure to high accelerations. When suction is applied to only one, that eye maintains clear vision while the other eye blacks out.

These experiments allow the conclusion that the loss of vision (blackout) that occurs without loss of consciousness during exposure to high acceleration is of retinal origin.

108. LAMBERT, E. H.
Physiologic studies of man's G tolerance in aircraft.
Federation Proc., *5*:59, 1946.

The author investigated G tolerance in a specially instrumented aircraft, maintaining a pattern similar to that used on a centrifuge for 10 to 15 seconds. He found dimming vision at 4.7G, loss of peripheral vision at 5.1G, and blackout at 5.4G. This was 0.7G higher than tolerance as passengers in aircraft and 1.4G higher than tolerance on centrifuge. G-suits afforded same increase in tolerance for pilots, passengers, and centrifuge subjects. Ear pulse, blood content of the ear, and pulse rate were similar in all three situations, but compensatory changes tended to occur 1 to 2 seconds earlier in aircraft.

109. LAMBERT, E. H., WOOD, E. H.
The problem of blackout and unconsciousness.
Medic. Clin. N. Amer., *30*:833, 1946. (Authors' Summary)

Before World War II engineering skill had increased the speed and maneuverability of airplanes to the point that their military effectiveness was limited by the inability of the pilot to withstand the forces developed in combat maneuvers. The sudden change in direction of flight which occurred in sharp turns and when pulling out of dives at high speed produced centrifugal force of such magnitude that the pilot was often rendered temporarily blind (blackout) or unconscious and unable to control his aircraft. It was recognized that if a pilot could be rendered resistant to the centrifugal force, he would have a tactical advantage over his adversary. This situation posed two problems for medical research; first, to determine the physiologic basis for the pilot's failure when exposed to centrifugal force and second, to develop means to improve his ability to withstand the force.

During the decade preceding the war the most significant research in this field was conducted in Germany. Relatively little was accomplished elsewhere. This is amply illustrated in Ham's review [90] of the reports on centrifugal force which had been published up to 1943. After the outbreak of the war, military and civilian laboratories devoted almost solely to the problem of centrifugal force appeared in quick succession in Canada, the United States, and Australia. As in other war projects, these laboratories conducted their investigations in secrecy. While the results of their researches have been gratifying both from a scientific and from a military point of view, little of the information obtained has yet been published in medical journals. It is the purpose of this paper to introduce the physician to some of the unique physiologic effects of centrifugal force on the human being and the means which have been devised to counteract these effects in the aviator.

110. LAMBERT, E. H., WOOD, E. H., BALDES, E. J.
Man's ability to withstand transverse acceleration when in the sitting position.
NAS–NRC–CAM–418, 1945.

Five subjects in a sitting position were exposed to transverse acceleration ($+G_x$) up to 10G for 3 to 10 seconds at a rate of onset of approximately 2G per second. Subjective sensations were experienced as follows: Pressure on chest and abdomen, not painful or distracting; dyspnea, particularly inspiratory, at high acceleration but not distracting in short runs; pain in epigastric region, sharply increasing with exposure, appeared between 4 and $7G_x$, and was decreased by elevating shoulders and hips to make back convex; dimming of vision occurred at 8 to $10G_x$ when head was elevated above level of heart; no complaints made with reference to head, legs, arms,

or back; movement of head and fingers possible up to $10G_x$.

Objective changes were as follows: Decrease in heart rate from 5 to 20 per min, increase in blood content of ear, no significant change in ear pulse, and premature systoles.

111. LEVERETT, S. D., CLARKE, N. P.
A technique for determining changes in force of cardiac contraction during acceleration.
Aerospace Med., 30:832, 1959.

Using an implanted strain gage sutured to the wall of the left ventricle, the authors exposed dogs to 3G at a rate of onset of 0.75G per sec for 15 seconds, and measured the contractile force of the ventricle. With onset of acceleration they observed initial decreases in both arterial pressure and force contraction. Six to 10 seconds later, the force increased to 25% above control, probably contributing to an associated rise in arterial pressure. Action was depressed by injection of dibenzyline, which reduces sympathoadrenal response to stress. With a gradual onset (1G per second), systolic pressure at head level fell to 0 between 5 and 6G, accompanied by gradually increasing force of contraction. With maintenance of 3G for 4 to 10 minutes the response was initially similar to that of rapid onset, but between 3 and 7 minutes after reaching peak there was a fall in pressure and decrease in force of contraction. The latter did not return to normal in the 10-minute observation period following centrifugation. Twenty-four hours later force was at control values.

112. LEVERETT, S. D., WHITNEY, R. U., ZUIDEMA, G. D.
Protective devices against acceleration.
In O. H. Gauer and G. D. Zuidema (eds.), *Gravitational Stress in Aerospace Medicine*, Little, Brown and Co., Boston, 1961.

113. LEWIS, D. H.
An analysis of some current methods of G-protection.
J. Aviat. Med., 26:479, 1955.

The author compared the effects of straining, the Navy Z–2 anti-G suit, and two types of experimental pressure suits on tolerance of four subjects to grayout during exposures to acceleration levels of $+2.5G_x$ to $+7.0G_x$. Control runs were made with the subjects unprotected and relaxed. Protection was observed as follows: straining, 1.1G; Z–2 suit, 1.2G; Z–2 suit plus straining, 2.2G; experimental pressure suit, 2.4G. For the Z–2 suit, a pressure of 3 psi per G above control tolerance level was required to produce protection; for the experimental pressure suit 2 psi per G was required. Discomfort of the experimental pressure suit limited tolerance.

114. LEWIS, D. H., DUANE, T. D.
Electroretinogram in man during blackout.
J. Appl. Physiol., 9:105, 1956.

The authors obtained a total of 136 observations of the electroretinogram (ERG) on five subjects during G_z acceleration exposure to grayout and blackout levels. Changes, although probably not too significant, were observed in the a-wave and the b-wave as the effect of acceleration on vision increased, ERG persisted during blackout. Collapse of retinal arterioles was observed during blackout. It was also noted that the consensual light reflex persisted. Persistence of the ERG was interpreted as indicating that rods and cones still react to light, although the subject cannot see. Since the consensual reflex persists during blackout, nerve transmission must continue. Also, the reflex pathways must be functioning at the level of the brain stem.

Initially, then, it would seem that the site of neural block in blackout is at some higher point than the nucleus, perhaps the visual cortex. Since, however, pressure on the eyeballs decreases the blackout threshold, and reduced extraocular pressure increases the blackout threshold, the retina appears more likely to be implicated. The visual and reflex pathways differ in that the former has an extra cell body and synapse in the superficial portion of the retina, namely, the cell body of the ganglion cell and the synapse between the bipolar and ganglion cells. These structures are supplied by branches of the central retinal artery which has been shown to collapse with onset of blackout. Thus, these structures are deprived of blood more than the deeper structures of the retina which are supplied by the plexus of vessels from the choroidal circulation. The authors therefore suggest that the block in blackout is at the level of the ganglion cell.

115. LINDBERG, E. F., MARSHALL, H. W., SUTTERER, W. F., MCGUIRE, T. F., WOOD, E. H.
Studies of cardiac output and circulatory pressures in human beings during forward acceleration.
Aerospace Med., 33:81, 1962.

Acceleration through the anterior-posterior axis of the body excites relatively little reflex activity in the cardiovascular system, since the vector of acceleration is perpendicular to the columns of blood. Visual or cerebral symptoms are rarely found with transverse accelerations below 20G. The authors investigated cardiac output, heart rate, mean aortic pressure, stroke volume, peripheral vascular resistance, and right atrial pressure in six subjects in the supine seated position during a total of 80 exposures to 2, $3\frac{1}{2}$, and 5G levels of forward acceleration for periods of up to 10 minutes. Results were widely variable.

Cardiac output: Higher in supine seated position than in upright seated position. No systematic change was found during 10-minute exposures to 2G. There was a statistically significant tendency toward higher cardiac output during exposures to 3.5 and 5G, but no progressive increase was observed during 10-minute exposures at those levels.

Heart rate: Higher during initial determinations

than during subsequent determinations at 1G following exposure to acceleration. No systematic change occurred during 10-minute exposure to 2G. A systematic increase was found at 3.5 and 5G, but no progressive changes during 10-minute exposure.

Stroke volume: Higher in supine than in upright position. Much variability, but a decrease was found during 5G exposure of 10 minutes' duration.

Mean aortic pressure: Higher in supine than in upright position. A consistent increase was recorded in all subjects upon exposure to forward acceleration.

Total peripheral resistance was represented by ratio of pressure to flow. Approximately same in upright as in seated position. Increased with exposure to 2G. Postacceleration control value was higher than preacceleration value and did not differ significantly from values obtained during exposure. Results suggest that the hemodynamic status of subjects was different during the first control determination of cardiac output. No systematic alteration in resistance occurred as exposure was prolonged. A slight increase at 3½ and 5G was followed by a postacceleration decrease.

Right atrial pressure: Increment in atrial pressure became greater as magnitude of acceleration increased, a fourfold increase taking place in some cases at 5G. A gradual decline from the high level attained at onset occurred as exposure was prolonged to 10 minutes. A systematic decrease postacceleration, as compared with preacceleration, suggested possible loss of volume of circulating blood, or increase in capacity of vascular bed, or both.

Comment: All changes are relatively small. This does not preclude the possibility that larger changes may take place above 5G. There are no overt signs of decompensation when acceleration is prolonged to 10 minutes. The most striking adjustments appear to occur with first exposure. Changes in cardiac output and heart rate, and increase in resistance, occurring during and after first exposure, may have been due to change in psychologic status of subject associated with completion of period of centrifuge rotation. All measurements were referred to midchest region at level of the third intercostal space, with no other adjustments to compensate for changes in heart level occurring with acceleration. The most dramatic effect was a uniform increase in the right atrial pressure, averaging 12 mm Hg at the onset of exposures to 5G. This was presumably the results of increased blood volume in the thorax together with anterior-posterior compression of thorax and abdomen. The decline in right atrial pressure as exposure was prolonged to 10 minutes may have resulted from an expanding vascular bed in dependent portions of systemic and pulmonary circulations not supported by counter pressure, and also from loss of plasma volume due to fluid shift from blood to extravascular space.

116. LINDBERG, E. F., SUTTERER, W. F., MARSHALL, H. W., HEADLEY, R. N., WOOD, E. H.
Measurement of cardiac output during headward acceleration using the dye-dilution technique.
Aerospace Med., 31:817, 1960.

Measurements of cardiac output, heart rate, stroke volume, mean arterial pressure, and systemic vascular resistance were made in six seated subjects in 51 one-minute exposures to 2, 3, and $4G_z$ levels in a centrifuge. Determinations were made during the period 20 to 40 seconds after onset of acceleration. Results were widely variable, but as compared with control showed decreases of 7%, 18%, and 22% in cardiac output, decreases of 24%, 37%, and 49% in stroke volume, increases in heart rate of 14%, 35%, and 56%, in mean aortic pressure of 9%, 21%, 27%, and in systemic vascular resistance of 17%, 41%, and 59%, during the 2, 3, and $4G_z$ accelerations, respectively. Note that these results refer to a period 20 to 40 seconds after onset of acceleration and not to a period of cardiovascular adaptation. Inflation of anti-G suit produced a significant difference only in mean aortic pressure, averaging 15 mm Hg above values without suit. Thus G suit protection is related to an increase in systemic vascular resistance, and not to an increase in cardiac output.

117. LINDBERG, E. F., WOOD, E. H.
Acceleration.
In J. H. U. Brown (ed.), *Physiology of Man in Space,* Academic Press, New York and London, 1963. (Authors' Summary)

The sequence of the changes that occur during sustained exposure to headward acceleration may be summarized as follows:

1. Acceleration increases the weight of the blood and tissues.

2. The increased weight of the blood reduces blood pressure at head level to such a degree that disturbances in vision and consciousness may occur.

3. The decrease in arterial pressure at head level initiates pressor reflexes that become effective in about 7 sec. The resulting increase in arterial pressure at heart level is usually sufficient to produce some degree of recovery at head level, even though the acceleration is continued. The heart rate starts to increase almost at the moment of onset of acceleration, and it increases progressively until the compensatory reactions occur.

After the compensatory rise in arterial pressure, some slowing of heart rate occurs. The maximal heart rate attained is usually proportional to the decrease in arterial pressure.

Hemodynamic studies, performed after these compensatory reflexes are in effect, reveal a moderate decrease in cardiac output, a pronounced decrease in stroke volume, and an increase in systemic vascular resistance, as viewed from the level of the heart. These conditions apparently prevail over long periods

of acceleration without signs of impending failure of the reflexes to maintain compensation.

Cerebral blood flow tends to be maintained in the face of decreased arterial pressure at head level by means of the negative pressure that prevails in the cranial cavity under this circumstance, coupled with the increase in the negative venous pressure recorded in the jugular bulb. This acts to preserve the arteriovenous pressure differential in spite of the decrease in arterial pressure at head level.

Headward acceleration produces alterations in respiratory physiology, as shown by the decreased lung compliance, decreased total vital capacity, and apparent disturbance in perfusion/ventilation ratio. Increasingly severe degrees of oxygen desaturation of arterial blood have been recorded in relation to the level of acceleration. The degree of arterial hypoxemia does not, however, appear to increase after the initial 1 to 2 minutes of exposure to a given level of acceleration.

118. LIPKIN, M., RATCLIFFE, H. L.
Some effects of cyclic acceleration on rhesus monkeys.
J. Aviat. Med., *25*:594, 1954.
NADC–MA–5404.

In this study, 18 rhesus monkeys (Macaca mulata) were subjected to acceleration forces of either 25G or 35G, which were combined with from 30 to 150 rotations per minute. Two monkeys were accelerated to 25 and 35G, then allowed to decelerate while being rotated at 110 turns per minute. All animals were sacrificed with intravenous Nembutal within 1 to 6 hours after exposure.

Postmortem examination revealed tissue damage in the internal organs of all animals exposed to this type of acceleration. The damage could be grouped in three categories: (1) Vascular congestion, edema, and hemorrhage; (2) formation of hyaline thrombi; and (3) separation of parenchymal liver cells.

A comparison is made with rhesus monkeys unexposed to acceleration, and with others exposed to positive and negative acceleration of 40G. It is suggested that the observed effects of cyclic acceleration are the result of more profound changes than can be accounted for on the basis of intravascular pressure rise due to the acceleration forces. It is recommended that the possible implications of these studies to man in high performance aircraft be given further study.

119. LIVINGSTON, B. C.
The problems of "blackout" in aviation (amaurosis fugax).
Brit. J. Surg., *26*:749, 1939.

The occurrence, clinical features, and operational significance of blackout in flight are discussed. The author notes that the retina is peculiar in that it is more dependent upon oxygen for the maintenance of vitality and function than any other tissue within the body. The oxygen demand represents a fourfold value over other structures. He points out that in determining the cause of blackout, not only must the retina and its blood supply be considered but also the visual pathway, the external geniculate body, the calcarine fissure, and the cortical association tracts. The author notes that oxygen has little effect in raising the blackout threshold, but carbon dioxide seems to be beneficial.

120. LIVINGSTON, R. B.
Cerebro-spinal fluid.
In T. C. Ruch and J. F. Fulton (eds.), *Medical Physiology and Biophysics*, W. B. Saunders Co., Philadelphia and London, 1960.

121. LOMBARD, C. F., ROTH, H. P., DRURY, D. R.
The influence of radial acceleration on respiration in human beings.
J. Aviat. Med., *19*:355, 1948.

Direct spirometry was undertaken on a small number of subjects exposed to $-3G_z$ and $+5G_z$, to measure tidal air, vital capacity, changes in volume of air in lungs at end of expiration, and respiratory rate. Results were as follows: (1) at $-3G$, slight increase in respiratory rate, decrease in tidal volume, decrease in lung volume at end of expiration, decrease in vital capacity, (2) at $+5G$ (without G suit or tight belt), slight increase in respiratory rate, increase in tidal volume, increase in lung volume at end of expiration, decrease in vital capacity, (3) at $+6G$ (with G suit), moderate increase in respiratory rate, decrease in tidal volume, decrease in lung volume at end of expiration, (4) at $+5G$ (with belt), slight increase in respiratory rate, increasing with G, slight increase in tidal volume, slight increase in lung volume at end of expiration.

Negative G findings suggest displacement of diaphragm toward head and/or pooling of blood in pulmonary circulation.

Decrease in vital capacity at $+5G$ is explained by pressure of weight of arms and shoulders on thorax, limiting maximum inhalation. Increase in lung volume at end of expiration is ascribed to shifting of abdominal viscera, diaphragm, and blood toward feet. At $+6G$ with G suit, the decrease in lung volume is believed due to suit's preventing displacement of viscera, etc. Increase in respiratory rate is perhaps a result of controlled respiration, but is accompanied by decreased tidal volume. Findings at $+5G$ with and without belt were similar; the belt made no appreciable difference.

122. MACIOLEK, J. A.
Circulatory reflex activity as a G-protective device.
WADC–TN–55–1, 1955.

Response of seven seated subjects to positive acceleration with rate of onset of 1G per second was compared with tolerance to runs with rate of onset from 0.07 to 0.1G per second. The approximate blackout

threshold for standard runs was 3.7G, while with slow onset blackout occurred at 6.2G. The author suggests that 2.5G difference is the measure of response of various mechanisms tending to sustain blood pressure in man under gravitational stress.

123. MARUKHANYAN, E. V.
Electrocardiogram changes and disturbance of the CNS under the influence of acceleration.
Sechenov Physiol. J. USSR, 47:36, 1961.

The author exposed subjects to $+7G_z$ for unstated duration, measuring ECG, arterial pressure, respiration, and other indices. The observed ECG change was compatible with moderate deflection of electrical axis to the right. Also noted were displacement of ST and change in T wave, most pronounced 10 to 20 seconds before disturbance in cerebral blood circulation, as indicated by visual disturbances and syncope. The ST and T changes continued during the disturbances. The author suggests that changes in ST and T warn of the possible appearance of deep functional disturbances of the central nervous system.

124. MCCUTCHEON, E. P., BERRY, C. A., KELLY, G. F., RAPP, R. M., HACKWORTH, R.
Physiological responses of the astronaut.
In Results of the Second United States Manned Orbital Space Flight, May 24, 1962.
NASA SP-6, 1962.

Acceleration of powered flight occurred in two phases: (a) During first 129 seconds from lift-off to booster-engine cutoff, and (b) during next 180 seconds. In the former, accelerations rose from +1G to +6.5G; in the latter they rose from +1.3G to +7.8G. Reentry acceleration peaked at +7.5G, with a duration of 3½ minutes from onset of initial +1G through peak to final +1G. Heart rate during launch increased from initial 84 per minute to 96 per minute, but maximum rate was not associated with maximum acceleration. Maximum heart rate at reentry (104) was at drogue parachute deployment. Mean rate during reentry was 84. The astronaut stated that the duration of reentry peak acceleration was longer than expected and that more forceful breathing was required to maintain speech. Landing impact was less severe than expected.

125. MCGUIRE, T. F., MARSHALL, H. W., NOLAN, A. C., LINDBERG, E. F., WOOD, E. H.
Comparison of changes in arterial oxygen saturation during transverse acceleration as indicated by ear oximetry and by direct photometry on arterial blood.
Paper presented at 32nd annual meeting of the Aerospace Medical Association, Apr. 1961.
Abstracted in *Aerospace Med., 32*:242, 1961.

Six subjects were exposed to 2, 3.5, and $5G_x$ for 10-minute periods in the astronaut position. Oxygen saturation was measured by the ear oximeter and also by simultaneous cuvette oximetry from the radial artery. Ear pulse, which indicated a critical decline in blood pressure at ear level during headward acceleration, did not show changes during transverse acceleration. An exponential drop in blood volume occurred during acceleration, ranging from 0 to 65%. Oxygen saturation by oximeter decreased rapidly during the first minute of exposure and then remained relatively stable during the rest of the 10-minute run. Average decreases in saturation were 8%, 10%, and 12% at 2, 3.5, and 5G respectively. Direct photometry on arterial blood showed a qualitatively similar pattern of decrease during exposure, but the magnitude of this decrease was less than that of ear oximeter. Arterial oxygen saturation levels below 85% by cuvette were found in some subjects during exposure to $5G_x$. These decreases could be prevented by breathing 99.6% oxygen. The discrepancy between ear oximeter and cuvette is believed due to retarded blood flow through the ear caused by acceleration, with consequent increased extraction of oxygen from the ear by local tissues.

126. MEEHAN, J. P.
Subjective end-points in acceleration.
In Reports on Human Acceleration, NAS–NRC–901, 1961.

Except for measurable end-points of visual loss in unconsciousness, acceleration tolerance is currently determined by subjective response of pain and voluntary termination, where the rate of onset is slow and duration of ride relatively long. When rate of onset is rapid and duration short, tolerance is usually determined by subjective response or inferred from pathological damage. The paper includes a table summarizing acceleration end-points.

127. MEEHAN, J. P., BRANDT, W.
Para-amino hippurate and endogenous creatinine clearances in positive acceleration.
Aerospace Med., 31:220–224, 1960.

In eight experiments with three subjects, exposure to a positive acceleration of 3G for 10 minutes did not demonstrate any consistent changes in PAH clearances or creatinine clearances from control values.

128. MILLER, H., RILEY, M. B., BONDURANT, S., HIATT, E. P.
The duration of tolerance to positive acceleration.
Aerospace Med., 30:360, 1959.

Eight subjects were exposed to 3, 3½, 4, 4½, and 5G in the $+G_z$ vector with rate of onset of 0.07G per sec. Runs were terminated by (1) fatigue or blackout, (2) appearance of cardiac abnormalities, or (3) attainment of an arbitrary time limit. All runs were initially without G suits, and some were repeated with G suits. Time limits were 1 hour at 3 and $3\frac{1}{2}G$, and 20, 10, and 4 minutes at 4, 4½, and 5G. A few subjects were exposed at 6G for 2 minutes with G suits. Data are shown in a table. At $3G_z$, the time

limit was attained in all cases except when acceleration was stopped in error. At $3\frac{1}{2}G_z$ fatigue was largely limiting, with durations from 40 minutes to full time. No blackout occurred. At higher limits blackout became the limiting symptom. Musculoskeletal pain was common at $2\frac{1}{2}G$ and above. The G suit raised the threshold at $4\frac{1}{2}G$ and above, in terms of duration. Large variation in pulse rate was noted; usually the rate increased at the start and later decreased.

Each subject, particularly after long duration, required from 1 to 3 hours to recover from postrun symptoms of malaise and dizziness. No ECG abnormalities were observed. At higher levels petechiae were common in dependent parts. The authors suggest that over a long period, circulatory embarrassment and blackout might occur, perhaps as a result of progressive decrease in circulating blood volume or from fatigue or adaptation of vasoconstrictor reflexes. No such evidence was seen. No permanent visual damage was observed, nor was any expected since retinas were not ischemic for more than momentary periods. No correlation was observed between pulse rate, physical fitness, blackout, or other factors.

129. NOBLE, R. L., TAYLOR, N. B. G.
Antidiuretic substances in human urine after hemorrhage, fainting, dehydration and acceleration.
J. Physiol., 122:220, 1953. (Authors' Summary)

Gilman and Goodman (1937) reported the presence of an antidiuretic substance in the urine of dehydrated rats. Evidence was presented that the antidiuretic substance was identical with the secretion of the posterior lobe of the pituitary gland. This work has been confirmed by others (Boylston and Ivy, 1938; Bundshuh and Kuschinsky, 1939; Hare, Hickey, and Hare, 1941; Ingram, Ladd, and Benbow, 1938). In contrast to these results, Walker (1939) found an antidiuretic substance in the urine of normal rats but was unable to confirm that it was increased after dehydration or absent after hypophysectomy. Noble, Rinderknecht, and Williams (personal communication) observed that when male or female human subjects were dehydrated by the exclusion of fluid from the diet, the urine contained an antidiuretic substance which they believed to be the posterior lobe hormone. No antidiuretic substance was found in the urine of subjects with a normal fluid intake. Similar findings have been reported for normal female subjects, both pregnant and nonpregnant (Teel and Reid, 1939). Heller and Urban (1935) have shown that the rat can excrete injected posterior lobe extract in the urine; there are similar reports for other animals (Heller, 1937; Larson, 1939). Considerable evidence has been presented by Brun, Knudsen, and Raaschou (1945, 1946), that the antidiuresis following postural fainting is due to the liberation of hormone from the neurohypophysis.

The experiments reported in this paper describe the presence or absence of an antidiuretic substance in the urine in a variety of conditions. A preliminary report of this work has appeared (Taylor and Noble, 1959), and our observations concerning the results of the intravenous infusion of vasopressin in man have since been confirmed by Burn and Singh Grewal (1951).

130. NOLAN, A. C., MARSHALL, H. W., CRONIN, L., WOOD, E. H.
The effect of forward ($+G_x$) acceleration on arterial oxygen saturation.
Physiologist, vol. 4, no. 3, p. 83, 1961.

Arterial oxygen saturation was monitored by ear oximetry and in radial artery blood in six subjects during 3 minutes at 2, $3\frac{1}{2}$, 5, and sometimes 6G in the $+G_x$ vector. Subjects breathed air, 99.6% oxygen, air at 40 mm Hg, and air during hyperventilation. Chest X-rays were made before and 30 to 50 seconds after 5G acceleration. Pressures were recorded in aorta, radial artery, right atrium, esophagus, and rectum. During air breathing, oxygen saturation decreased with increasing G, beginning approximately 30 seconds after onset, and at 5G reached 87.3% about 80 seconds later. When acceleration ceased, return to control value occurred over about 70 seconds; at 5G the return was often incomplete, being up to 3% lower than control. During oxygen breathing, decrease was prevented or onset delayed and magnitude of desaturation less, 95.5% being the lowest figure at 5G. Progressive increase in right atrial pressure occurred with increasing G, reaching a mean of 32 mm Hg (3 times control) at 5G. Esophageal pressure was similarly increased to a lesser mean of 19 mm Hg at 5G. Authors suggest that oxygen desaturation is due to blood flow past atelectatic alveoli in dependent portions of lung, from increased segmental blood volume and pressure, plus increase in intrathoracic pressure. Atelectasis was demonstrable by X-ray after exposures to $5G_x$.

131. PETERSON, R. L.
Personnel seating research for Air Force aerospace vehicles.
SAE–751C, Apr. 27–30, 1964.

The Air Force Flight Dynamics Laboratory net seat research program is discussed. The net crew seat prototypes that were evaluated provided excellent body support during 1G comfort studies and centrifuge exposure up to 16.5G. However, undesirable rebound of seat occupant occurred during low-frequency vibration and ground-landing impact experiments. An experimental net seat system, described in the report, was designed to eliminate occupant rebound without compromising comfort and sustained acceleration support.

Prototype 16G aft-facing passenger seats utilizing the net body-support approach were designed, dynamically evaluated, and finally rejected because of excessive weight and failure to meet strength criteria.

Several seating configurations utilizing the net seat technique for body support are discussed.

132. PETTITT, J. A.
Multiple psychophysiologic measures during gradual onset acceleration.
WADC–TN–57–234, 1958.

Using $+G_z$ peaks of 2, 4, and 7 or blackout, the author exposed 15 trained subjects at a rate of onset of 1G per 15 seconds. An increase was observed in central nervous system arousal as measured by skin resistance changes with increase in G level. Those blacking out at higher G showed less arousal at the beginning and during the course of acceleration. The author suggested that blackout is not only dependent on cardiovascular system response, but is related to psychologic and neurophysiologic factors.

133. PINC, B. W., BARR, N. L.
Some responses of squirrel monkeys to high-G brief duration acceleration profiles.
SDCOR–TR–63–103, Apr. 1963.

It may be possible for organisms to survive much higher accelerative forces for much longer periods of time than had previously been thought possible. An articulated centrifuge was developed and 18 squirrel monkeys were placed on it and exposed to unconventionally high accelerative forces. Thirteen tests above $+300G_x$ over approximately 20-second time bases were conducted, of which eleven exceeded 400G. One death and some injuries occurred, and a typical general response, characterized by cardiovascular and neurologic signs, was observed. The mechanics of injury are discussed, as are six cases of electrocardiac arrest coincident with peak acceleration. Additional work is suggested.

134. POLIS, B. D.
Hormonal determinants of mammalian tolerance to acceleration stress.
J. Appl. Physiol., 16:211, 1961.
NADC–MA–6025, 1960.

The author notes that acceleration investigations have been dominated by hemodynamic concepts but that at the cellular level physiological stress may be defined in terms of substrate supply and energy demand, and that under conditions of limited supply the distribution of energy available could be controlled by hormonal mechanisms. To define these factors he exposed rats to acceleration stress of +20G, one group having undergone hypophysectomy and another group adrenalectomy. Using as a physiological end point the time to reduce the heart rate from 8 to 2 beats per sec, he demonstrated a significant increase in the survival time of the former group (300%) and a significant loss (60%) in the latter group.

Hypophysectomy is normally followed by atrophy of the adrenal cortex. This was demonstrated by a gradual loss in the ability of hypophysectomized rats to resist acceleration stress, although at 15 weeks after the operation the increase was greater in the hypo group than in the control group. In contrast, after adrenalectomy, the survivors showed an enhanced tolerance to acceleration stress. When both operations were performed, the rats approximated the normal period.

Hypophysectomy is normally associated with loss of tolerance, for example, to heat, cold, disease, and fatigue. The author argues, however, that limiting factors for survival reside in the efficiency in which energy is made available to the cell from respiratory enzymes. He believes that in the normal animal the maximum energy available is distributed by hormonal factors among various demands for this energy. Some is expended in long term needs, as for protein synthesis, fat conservation, and glycogen storage, and some is available for immediate use in muscle contraction, nerve conduction, osmosis, etc. The author suggests that hypophysectomy depresses long-term demands and allows vital cellular function; thus, in hypophysectomized animals a demand for elementary survival over a short term is more readily met because less energy is diverted for long-term needs.

135. POLIS, B. D.
Increase in acceleration tolerance of the rat by 2-dimethylaminoethyl p-chlorophenoxyacetate.
NADC–MA–6136, 1961.

On the basis that acceleration tolerance of rats was improved by hypophysectomy and reduced by adrenalectomy, the author investigated chemicals to achieve the same end pharmacologically. Lucidril, the dimethylaminoethyl ester of parachlorophenoxyacetic acid was found to increase significantly the tolerance of rats to acceleration of 20G. Median survival time increased almost threefold over control survival time of 12.5 minutes. The effectiveness of the drug persisted for a period of 4 hours after injection. A latent period of 3 to 4 days' treatment seemed necessary before tolerance became apparent. The activity of the drug was dose dependent, with no changes at 50 mg, significant increases at 75 mg, and greater increases at 100 mg. The drug action appears to be mediated via the hypothalamic area of the brain, facilitating mechanisms for maintaining blood pressure and hence cerebral circulation. Alternatively, because of its relationship to acetylcholine, it may potentiate cholinergic reactions under hypoxic conditions such as acceleration stress. The drug has low toxicity, and has been used in humans suffering cerebral injury with some dramatic results.

136. POLIS, B. D., SHMUKLER, H. W., CHIANTI, M.
Changes in the "amino acid composition of rat brain" caused by acceleration stress.

Paper presented at 33rd annual meeting of the Aerospace Medical Association, Apr. 1962.
Abstracted in *Aerospace Med.*, *33*:349, 1962. (Authors' Summary)

This study was undertaken in an effort to reveal the metabolic defects in cerebral metabolism induced by acceleration. Rat brains were rapidly excised from normal and centrifuged animals, frozen, weighed, deproteinized with picric acid, and analyzed for the complete free amino acid pool composition by ion exchange chromatography. Large decreases (> 50%) were found for the amino acid B-hydroxyaspartic acid, as well as for serine, urea, and glutathione. A large increase in the concentration of free ammonia was also found. The interrelationships of the changes in amino acid composition suggest a block in the energy-yielding mechanisms from the respiratory enzyme systems in mitochondria.

137. POPPEN, J. R.
Report on the physiological effects of sudden changes in the speed and direction of airplane flight.
Dept. of Physiology, Harvard School of Public Health, Boston, 1932.

138. PRESTON-THOMAS, H., EDELBERG, R., HENRY, J. P., MILLER, J., SALZMAN, E. W., ZUIDEMA, G. D.
Human tolerance to multistage rocket acceleration curves.
J. Aviat. Med., *26*:390, 1955.

The authors exposed subjects to simulated rocket launch with peaks of $+8G_x$, $+5.8G_x$, and $+5.8G_x$ over a period of 6 minutes in the supine position with back raised at 15° and legs at 60°. The subjects performed a tracking task. Runs were subjectively tolerable with complaint of fullness in chest and difficulty in breathing. There was no difficulty in control movements. Vertical nystagmus occurred in some during the later part of the deceleration phase, and varied in severity from subject to subject. Performance deteriorated slightly to negligibly.

139. REED, J. H., JR., BURGESS, B. F., SANDLER, H.
Effects on arterial oxygen saturation of positive pressure breathing during acceleration.
Aerospace Med., *35*:238, 1964. (Authors' Summary)

Twenty-two centrifuge runs were performed on eight subjects in whom arterial oxygen saturation was continually monitored by means of a Waters cuvette while the subjects were exposed to various transverse acceleration $+G_x$ at a seat angle of 6° head up. These runs were made during conditions of breathing air (control), air at positive pressure, pure oxygen (control), and pure oxygen at positive pressure. The positive pressure was metered automatically to provide 3 mm Hg per G above ambient pressure.

The results of this experiment show that the slope of the curve of oxygen saturation plotted against time for air and air at positive pressure decreased approximately 3% every 10 seconds, beginning 10 to 20 seconds after the onset of the acceleration.

During the oxygen breathing studies, a lowering in arterial oxygen saturation was observed approximately 100 seconds after the onset of acceleration.

A method is suggested as a means of estimating physiological limits for theoretical profiles of acceleration G plotted against time.

140. ROGERS, T. A., SMEDAL, H. A.
Ventilatory advantage of backward transverse acceleration.
Aerospace Med., *32*:737, 1961.

Four subjects were exposed to 4, 6, and 8G in the $\pm G_x$ vectors with open-loop operation of the Johnsville centrifuge. Volume changes were recorded by using a WEDGE spirometer and included tidal air, minute volume, inspiratory capacity, expiratory reserve, and vital capacity at the two lower G levels. At $\pm 8G_x$, tidal volume and minute ventilation alone were recorded. Results are displayed in a table. In the $-G_x$ position, vital capacity is slightly reduced up to 6G, but in the $+G_x$ position, vital capacity is greatly decreased at the same level. With $+G_x$ acceleration, minute ventilation was well maintained up to 8G; with $-G_x$, minute ventilation increased. Subjectively there is a strong drive to hyperpnea during $-G_x$ acceleration, borne out by increased minute volume, but since drive can be achieved dyspnea is not as severe as during $+G_x$.

The cause of drive may be cortical, or it may result from tonic stimulation of lung or chest wall receptors sensitive to distortion. These receptors would be stimulated in either vector, but with $-G_x$ compliance remains low enough for the subject to respond. Increased drive could be due to hypercapnia from decreased alveolar ventilation. The latter is partially compensated, however, by decreased functional residual capacity, and also it is unlikely that hypercapnia could have developed in some of the very short (15 second) runs at 8G where it was also experienced.

141. ROSENFELD, S., LOMBARD, C. F.
Cardiovascular pressor reflex mechanism and cerebral circulation under negative G head-to-tail acceleration.
J. Aviat. Med., *21*:293–303, 1950.

Data from experiments with various animals, including 10 goats, 10 dogs, 5 monkeys, and 3 rabbits, compared with data available for man, indicate that the monkey and the dog respond in a manner which most closely parallels the reflex cardiovascular response elicited in man during negative G. The goat does not simulate this response pattern. The major changes to cephalic circulation during negative acceleration, as measured from the carotid artery and external jugular vein connected to Statham

strain gages at the level of the brain, include the following.

At the onset of radial acceleration the arteriovenous pressure difference in man, monkey, and goat is slightly increased, but in the dog it is slightly decreased. During radial acceleration of 15 seconds' duration, the arteriovenous pressure difference diminishes gradually in man (30%), monkey (12%), and dog (65%), but increases in the goat (65%), and is associated with a bradycardia of various degrees. At the termination of radial acceleration, the arteriovenous pressure difference drops markedly within 1 to 4 seconds in man (65%), monkey (40%), and dog (60%), and drops to the normal prerun level in the goat.

It is believed that at the termination of acceleration the animal or subject experiences the most harmful insult to cerebral circulation. Factors which tend to maintain minute cardiac output, as well as factors preventing generalized vasodilatation, should be investigated for possibilities of offering greater tolerance to negative acceleration.

Finally, it seems likely that the symptoms experienced by pilots exposed to negative G may be attributed to two phenomena: (1) the increase in intravascular and extravascular cephalic pressure, and (2) the stagnation of cerebral flow. The former produces the feeling of extreme fullness of the head, while the latter is probably responsible for factors of mental confusion, fainting, and unconsciousness. However, the possibility of a central type of syncope from stimulation of the carotid sinus cannot be overlooked.

142. RUFF, S., STRUGHOLD, H.
Grundriss der Luftfahrtmedizin.
J. A. Barth, Leipzig, 1939.

143. RUSHMER, R. F., BECKMAN, E. L., LEE, D.
Protection of the cerebral circulation by the cerebrospinal fluid under the influence of radial acceleration.
J. Physiol., 15:355, 1947.

Using an elegant technique, the authors made simultaneous recordings of venous and cerebrospinal fluid pressures in anesthetized cats at the vertex of the skull and at neck level under the effects of $+6G_z$ to $-6G_z$. They also measured changes in carotid and jugular pressures during exposure to negative G. Each animal had two runs of 15 seconds' duration at 2, 3, 4, 5, and 6G in the $-G_z$ vector. Cerebrospinal fluid pressures ranged from 80 cc CSF above normal to 190 cc CSF below normal. Throughout this range CSF and venous pressures measured at the same level varied together and by approximately the same amount ($r = 0.91$). CSF and venous pressures remained relatively unchanged at or near heart level during exposures to either positive or negative G.

Findings were interpreted to indicate that columns of CSF and blood producing these pressure changes must be of approximately the same length, or alternatively, that CSF pressure is a reflection of local venous pressure. Because of anastomoses, so long as no pathologic changes occur, a difference in pressure between CSF and venous pressure would result in expulsion or retention of blood within venous reservoir or plexuses so that equilibrium could be maintained. The authors consider that veins, and probably minute vessels, are almost perfectly protected against sudden changes in intravascular pressure by simultaneous changes of the same magnitude in CSF pressure. In the majority of cases, arterial blood pressure at neck level increased by larger increments than the venous pressure at some time during exposure to negative G. Thus cerebral arteries are afforded considerable but incomplete protection under conditions of acceleration.

144. RYAN, E. A., KERR, W. K., FRANKS, W. R.
Some physiological findings on normal men subjected to negative G.
J. Aviat. Med., 21:173, 1950.

This represents the first definitive study of negative G and was carried out on more than a hundred subjects during the year of 1943–1944. Investigations included tilt-table tests, −1G for 1 minute, and centrifuge tests to $-3G_z$ for 5 seconds through a total duration of 25 seconds. The subjects were seated on the centrifuge with line of back 30° below horizontal, legs and thighs vertically up. Thus a gradient of G occurred, depending on where measured; for example, for $-3G_z$ the cam gradient varied from −0.175 to −3.28.

The authors demonstrated that a feeling of pressure in the head region is the outstanding symptom on exposure to $-3G_z$. Visual symptoms are common, consisting of blurring, graying, or reddening. Negative G produces slowing of the pulse rate in proportion to the magnitude of acceleration. While 93% of slowing occurs within the first 3 seconds, maximum slowing is not attained until 10 to 15 seconds. This is followed by significant and partial recovery above the lowest level until equilibrium pulse rate is reached within 25 to 45 seconds after onset. These changes may be mediated through carotid sinus reflex. Thus initial bradycardia occurs through vagal reflex; meanwhile alterations occur in caliber of blood vessels, with redistribution of circulating blood, mediated through the sympathetic nervous system. Thus, resulting pressure in carotid sinus is relatively decreased over initial value, and consequently pulse rate speeds up. Pulse rate quickly returns to normal or a little above on cessation of negative G. A more marked slowing of pulse rate occurs on last exposure of series as compared with first, either on one day or successive days. This change is correlated with an acquired subjective increase in tolerance to negative G.

ECG's taken under negative G show changes more marked as negative G increases. Most striking are prolonged periods of cardiac asystole. No subjective cardiac symptoms occurred. ECG changes are simi-

lar to those resulting from pressure on sensitive carotid sinus.

145. SADOFF, M.
Effects of high sustained acceleration on pilots' performance and dynamic response.
NASA TN D–2067, 1964.

A study was conducted by Ames Research Center on the human centrifuge at the U.S. Naval Air Development Center, Johnsville, Pa., to determine the effects of sustained high acceleration on pilot control capabilities. The results showed that the predominant effect of acceleration stress was an increased attenuation of the pilot's dynamic response and an associated large increase in his errors at the higher frequency components in the task command input function.

This impairment of the pilot's control capability suggests that it may not be desirable to impose precise attitude-stabilization tasks on human pilots when high-frequency control during periods of high sustained accelerations is required of him. Results of the present study indicate that for control frequencies above about ½ cps and at acceleration levels above about 6G, an appreciable deterioration in pilot attitude-control performance can be expected.

146. SEM-JACOBSEN, C. W.
Electroencephalographic study of pilot stressed in flight.
Aerospace Med., 30:797–801, 1959.
(Abstract from STL–9990–6302–KU–000)

Eight-channel EEG tracings were obtained from 30 experienced military jet pilots in order to determine: (1) The effect of combat flight stress on pilot consciousness level, (2) whether the brain is stressed under combat flight conditions to such a degree that this stress can be measured by airborne EEG, and (3) whether there is a correlation between the pilot's in-flight EEG, his ability to fly an interceptor, and his accident rate due to pilot error.

The flight conditions were a composite of 10 maneuvers common to combat interceptor flight. EEG tracings were compared with pilot flight performance. The feasibility of EEG as an in-flight determiner of physiologic response to flight stress is considered.

147. SEM-JACOBSEN, C. W.
Recording of in-flight stress in jet fighter planes.
Aerospace Med., 31:320, 1960.

EEG's, in-flight movies, and in-flight records of maneuvers were taken on 40 missions in high-performance aircraft operated by pilots assessed as being fit. Results divided the pilots into three groups: (a) minimal EEG changes, with no effect observed in the movies, (b) short episodes of high-voltage delta-theta activity on EEG, and 8-second convulsions on the movie, and (c) in one pilot, major EEG abnormality and unconsciousness for 30 seconds.

148. SEM-JACOBSEN, C. W., NILSENG, O., PATTEN, C., ERIKSEN, O.
Electroencephalographic recording in simulated combat flight in a jet fighter plane.
EEG and Clin. Neurophys. J., 11:154, 1958.

Using a transistorized EEG with eight channels, the authors obtained in-flight EEG records under combat-type maneuvers. The EEG unit was largely unaffected by the environment. "Great changes" were seen during some maneuvers and marked slow waves appeared during the landing pattern.

149. SEM-JACOBSEN, C. W., SEM-JACOBSEN, I. E.
Cerebral activity measured by EEG and flight performance in 25 jet fighter pilots during flight stress.
GAUSTAD–6702–FTR, 1961.

Airborne EEG's were taken on 25 selected pilots. Ten of these had excellent flight records and showed minimal EEG change. Twelve pilots had a record of pilot-error accident. Seven of these showed significant changes in the EEG during flight. None of the pilots who showed EEG changes had an excellent flight record. Three of those who showed significant EEG changes in flight showed only minimal changes under a heavier G-load in the centrifuge.

150. SHAW, R. S.
Negative acceleration.
Military Surgeon, 102:483, 1948.

On exposure to $-3G_z$, superficial venous pressure rises to about 100 mm Hg, with rapid fall off on cessation of acceleration. An initial peak of arterial pressure may be seen, probably from reflux of blood from arteries of caudal portion of body. This is followed by bradycardia and slow fall of pressure, probably from carotid sinus effects. These effects are not seen in animals with denervated carotid sinus. Marked vagal effects, such as partial or complete heart block and ventricular extrasystoles, are seen in ECG with higher acceleration. Edema and rupture of vessels may occur in small vessels of conjunctiva and accessory sinuses. Intracranial vessels remain uninjured, probably because of protective effects of cerebrospinal fluid pressure. Rushmer et al.[143] (1947) showed that up to $-6G_z$, venous pressure and CSF remained equal. Retinal or eye damage is not seen, although a redout is occasionally observed, probably from upward movement of conjunctiva of lower lid.

With high acceleration (ejection), there is a rapid displacement of the diaphragm which may increase intrathoracic pressure (180 mm Hg at $-7G_z$). With still higher acceleration (drop tests), damage to diaphragm, viscera, and mesentery may occur. Extremely high acceleration may produce fracture, especially at the clavicle, usually the point of support (opening shock).

Minimal accurate data on tolerance are available. Paper includes useful tolerance curves, providing

information on (a) $-G_z$ loads causing injury to vessels of head, (b) tolerable downward seat ejection, (c) tolerable sustained negative acceleration, (d) impact shock data.

Protection is aimed at reducing venous pressure in the frontal veins. Slight protection is gained from 45° and 60° partially supine position, and slightly greater protection from similar partially prone position. All straining maneuvers must be avoided. Shoulder harness will increase venous pressure about 5 mm under negative G, up to $-3G_z$. The Muller maneuver is the inverse of a valsalva, that is, closing the glottis and attempting inspiration. This will result in a pressure decrease of about 30 mm Hg in the veins of head, and will afford relief up to $3\frac{1}{2}G$. It gives rise to dyspnea, cough, and substernal pain occasionally. Counterpressure by facial mask will produce relief, again up to about $-3G_z$.

151. SIEKER, H. O.
Devices for protection against negative acceleration. Part I. Centrifuge studies.
WADC–TR–52–87, 1952.

Human subjects were exposed to negative acceleration with and without protection. The tolerance limit for negative acceleration was 2.5G for unprotected subjects in upright seated position. Tolerance was limited by discomfort and bradycardia noted in ECG. Conjunctival hemorrhage and cardiac asystole were noted at 3G. With counterpressure, tolerance was increased to 5G. When applied in prone position, negative acceleration was limited to 4G.

152. SLONIM, A. R.
Effect of acceleration on blood creatine, creatinine and inorganic phosphorus in man.
J. Appl. Physiol., 15:271–274, 1960.

Ten healthy male subjects were centrifuged front to back at 6G for 3 minutes, with head and trunk inclined 25° forward in the direction of acceleration. Average control values of blood creatine, creatinine, and inorganic phosphorus were almost identical to those found immediately after exposure to the acceleration. Relatively large differences (test minus control) in some individuals were not significant. Acceleration of the magnitude and/or duration used in this study does not appear to be comparable to the effects of vigorous exercise on the phosphocreatine system of man.

153. SLONIM, A. R.
Effects of relatively high accelerations on some biological systems.
J. Appl. Physiol., 16:221–225, 1961.

Fasting male subjects were exposed to forward accelerations (12° back angle) at fatiguing levels, varying both in amplitude and duration, to accelerations at nonfatiguing levels and mock runs, and to treadmill exercise. The following analyses were made: plasma bicarbonate; blood glucose, phosphorus, and creatinine; urine creatinine; urine volume; urinalysis; and estimate of creatinine clearance. The only consistent change noted after fatiguing accelerations was a small rise in blood creatinine ($P > 0.05$). Exercise, however, resulted in a marked ($P > 0.01$) decrease in bicarbonate, rise in blood creatinine, and drop in clearance. Accelerations in comparison with exercise showed little effect on either muscular or renal activity. No correlation was found between any of the biochemical measurements and acceleration intensity, nor were any differences noted between real and mock accelerations, indicating that none of these tests could serve as an index of the severity of accelerative stress. The fatigue associated with high accelerations is not easily explainable in terms of increased muscular activity.

154. SMEDAL, H. A., CREER, B. Y., WINGROVE, R. C.
Ability of pilots to perform a control task in various sustained acceleration fields.
Aerospace Med., 31:901, 1960.

The authors describe criteria for tolerance as magnitude of accelerative force, rate of onset, direction in which applied, and duration of performance capability. Six subjects carried out a complex tracking task on the AMAL centrifuge while exposed to different levels and durations of G. Protocol is not stated. Results are recorded in a table in terms of magnitude, tolerance time, and position (EBO, EBD & O, EBD). Tolerance varied from 5.7 to 7G and $1\frac{1}{4}$ to $6\frac{1}{2}$ minutes. Time-tolerance boundaries were constructed and are illustrated in a figure, which also shows accelerations anticipated during reentry.

It was demonstrated that, with suitable restraint, tolerance to EBO was as good as to EBI. In entry from parabolic and lunar velocity, man's tolerance to acceleration as determined here could be exceeded. Tolerance was limited by visual, cardiovascular, and respiratory problems. During EBO, transient changes in visual acuity occurred, perhaps from distortion of corneal surfaces (probably tears). The authors suggest possibly lens displacement or tilting of retinal receptors. Loss of acuity was never critical. In EBD accelerations, graying or blackout occurred. Impairment of the cardiovascular system was manifested in petechial hemorrhage, tissue fluid accumulation, and transient arrhythmia. Hemorrhages occurred in the lower extremities in all EBD acceleration, as did tissue fluid accumulation. These symptoms also occurred in forearms on EBO acceleration and could be minimized by elastic bandages. Premature beats were frequently seen in EBD and EBI but never in EBO. Changes in S–T segment and elevations in amplitude of T waves occurred. All subjects agreed that respiration was less difficult during EBO and most difficult during EBI.

155. SMEDAL, H. A., ROGERS, T. A., DUANE, T. D., HOLDEN, G. R., SMITH, J. R.
The physiological limitations of performance during acceleration.
Aerospace Med., *34*:48, 1963.

The authors exposed subjects to accelerations up to 8G in the $+G_x$ and $-G_x$ vectors for unstated durations to further elucidate the differences between them. The investigation included visual, cardiovascular, respiratory, and performance studies. Visual equipment included a phoropter with Snellen chart and astigmatic dial, mounted on the centrifuge, and a Placido disk reflected in the cornea and photographed. Cardiovascular studies included blood pressure, ear pulse, ECG, and arterial oxygen saturation recorded by ear oximeter. In respiratory studies the Ames monitoring equipment was used with a bite mouthpiece rather than the face mask.

Results showed that during $+G_x$ acceleration, alveolar ventilation and arterial saturation are severely diminished. Indirect evidence, namely, graying of vision and decline in ear pulse amplitude, indicates that diminished venous return contributes to the progressive hypoxia and hypercapnia. Dyspnea is chiefly due to hypercapnia but also to deflation of lungs. Under $-G_x$, alveolar ventilation and arterial saturation are essentially normal. Ear pulse, blood pressure, and heart rate all suggest that cephalic flow is as good as during $+G_x$ acceleration or better. Visual findings showed no distortion of Placido disk compatible with corneal displacement, but a different and gross distortion with lacrimation. Similarly, acuity was reduced with lacrimation. The phoropter indicated that a double image could not be fused under acceleration. Blurring of vision occurred at $-6G_x$ without lacrimation, and graying occurred at $-8G_x$. The two may be related and associated with retinal ischemia. Among other respiratory findings, the nitrogen closed-circuit content was observed to rise on cessation of $+4G_x$ or $6G_x$. This may be due to return of previously pooled venous blood to pulmonary circulation, or to subsequent release of air trapped in compressed and unventilated portions of lungs.

Tracking performance under $-G_x$ did not show the clear-cut superiority to be expected from physiological advantages. Performance decrements were probably due to some other factor fortuitously similar, such as increase in intraoptic pressure or simply tears. Despite greater respiratory comfort, $-G_x$ acceleration is disliked, perhaps because of the restraint system.

156. SMEDAL, H. A., STINNETT, G. W., INNIS, R. C.
A restraint system enabling pilot control under moderately high acceleration in a varied acceleration field.
NASA TN D–91, 1960. (Authors' Summary)

A restraint system is described which was used in a joint centrifuge program by the Ames Research Center of the National Aeronautic and Space Administration and the Aviation Medical Acceleration Laboratory of the Naval Air Development Center. The program was designed to study the ability of a pilot in a forward-facing position to control an entry vehicle which employed lift. The pilot was required to carry out a relatively complex tracking problem on a flight simulator which involved the centrifuge operated as a closed-loop system. Dynamics typical of an entry vehicle were used and the pilot was subjected to varied acceleration-time profiles with relatively high accelerations, up to 7G, from various directions for approximately 2 to 5 minutes. In order to conduct these tests, it was necessary to design a special restraint system. This system combined a modified NASA posterior mold or couch with an anterior restraint made from nylon straps and nylon netting. A special support for the head and face was also incorporated into the restraint system. The use of this restraint system permitted a thorough study of some of the control problems of entry vehicles.

157. SNYDER, R. G.
Human survivability of extreme impacts in free fall.
CARI document 63–15, Federal Aviation Agency, Oklahoma City, 1963.

158. SQUIRES, R. D., JENSEN, R. E., SIPPLE, W. C., GORDON, J. J.
Electroencephalographic changes in human subjects during blackout produced by positive acceleration.
NADC–MA–6402, Apr. 1964.

Each of 13 human subjects was subjected alternately to a set of peak accelerations of 6 and 7G on two separate occasions. Peak G was attained in approximately 30 seconds after the initiation of a symmetrical, sinusoidal acceleration profile.

One channel of EEG was recorded by placing two active electrodes, one on the right and one on the left side of the calvaria, approximately 2 cm above and lateral to the occipital protuberance. The indifferent electrode was placed over the forehead in the midline. Analysis of the taped EEG signals was accomplished by a 14-channel continuous frequency analyzer using band-pass filters manufactured by Epsco. The voltage output of each band-pass filter was rectified and passed through a smoothing filter in order to obtain a direct-current voltage proportional to the amplitude of the frequency band passed by each filter.

Frequency analysis showed characteristic changes during visual grayout and blackout. An increase in beta frequencies (16 to 36 cps) occurred which showed the same general amplitude pattern as the acceleration profile.

The lower beta frequencies (16 to 19 cps) have a tendency to level out or to decrease during each

blackout, coincident with an increase in the lower frequency components. The alpha frequencies (8 to 13 cps) often appeared during grayout and blackout, but may disappear during very deep blackout when bursts of high-amplitude low-frequency components appear. The best index of the level of consciousness appears to be the inverse relationship between the depth of blackout and the amplitude of EEG frequencies in the range of 5 cps. The lower delta frequencies were not used since artifacts due to electrode displacement resulting from head movement were seen most frequently in this range of frequencies. Moreover, the 5 to 7 cps frequency band is associated with cerebral hypoxia which occurs during positive acceleration. This frequency band was also shown to be related to performance of specific performance tasks.

159. STAPP, J. P.
Problems of human engineering in regard to sudden decelerative forces on man.
Military Surgeon, 103:99, 1948.

160. STAPP, J. P.
Effects of mechanical force on living tissues. I—Abrupt deceleration and windblast.
J. Aviat. Med., 26:356, 1955.

161. STAPP, J. P.
The "G" spectrum in space flight dynamics. Lectures in Aerospace Medicine, School of Aviation Medicine, Brooks AFB, 1961.

The author discusses the techniques and expected acceleration in attaining orbital and escape velocities. He quotes the work of several others in delineating human tolerances, as follows.

Accelerations to orbital and escape velocity: Gauer and Ruff (1939) exposed human subjects to 11G for 3 minutes in transverse, front to back, or supine position without reaching tolerance. Bührlen (1937) exposed a human subject to 17G for 4 minutes, chest to back. Ballinger (1952) exposed seven subjects, supine, to 8G for 2 minutes 40 seconds, with resulting severe substernal pain and dyspnea; elevation of knees to 20° and trunk and head to provide eye-to-heart vertical distance of 7 inches allowed two out of three subjects to tolerate 10G for 2 minutes 6 seconds. Bondurant and Clarke (1958) confirmed dyspnea and chest pain, in the transverse vector, at 8G. With trunk angle greater than 70° to direction of acceleration, tolerance is limited to 7G by anterior chest pains. Decreasing below 70° increases blackout at lower accelerations. Best tolerances were found when leaning in direction of acceleration at 65° to 70° with legs and trunk elevated only 20° (Ballinger, 1952). Respiration was difficult above $4G_x$, and at 6 to $8G_x$ tolerance time was dependent on ability of subject to force abdominal breathing. Following exposure to 6 to $8G_x$, an unsteady gait with dizziness, vertigo, and occasional nausea persisted for 1 to 5 minutes after the run. Transverse acceleration back-to-front is limited by distribution of pressure on the body against straps and by hydrostatic pressure and vascular distention in legs. Tolerance limit is $5G_x$ with legs extended, and $8G_z$ when seated upright with legs at 90°. In positive G position, with the seat tilted back 13° and legs partially extended, tolerance is limited to 10 minutes at less than 4G. At 4 to $5G_z$ blackout is frequent. An occasional episode indicating imminent syncope occurred without relation to magnitude and duration. Disturbance often persisted several hours after episodes.

Performance of tracking task was possible with small loss of accuracy by nine subjects during three-stage peaks of $8G_x$, $5.8G_x$, and $5.8G_x$ for a total exposure of 6 minutes, with back and head 15° from horizontal and knees at 60° angle in transverse front-to-back direction (Preston-Thomas et al., 1955). Arm and leg movements were not effective above 6G. Wrists and fingers were movable in all positions in all magnitudes. Exposure to three-stage acceleration with rates of onset from 0.1 to 8G per second, to peaks of 8, 10, or $12G_x$, showed that subjects could see, think, and exercise at least finger control despite loss of peripheral vision, difficulty in breathing, and pain. Accuracy of coordination and competency of judgment were not evaluated.

Bondurant and Clarke investigated tolerance for duration of acceleration of 6, 8, 10, and $12G_x$ in semisupine subjects immersed in a tank of water. A 35° angle of trunk to line of force was optimal. Petechiae did not occur even at 12G, and freedom of movement was present regardless of magnitude. Free movement of the head increased likelihood of vertigo. Clark and Hardy (1959) investigated tolerance in the "iron maiden," where one subject withstood 31G for 5 seconds with minor nondisabling effects. Immersion during acceleration can approximately double tolerance limits if required.

Reentry acceleration: The Russian 4.5-ton space vehicle accomplished reentry in 1960 without exceeding 10G deceleration. Clarke et al. (1959) evaluated human response to rearward-facing reentry acceleration. Four subjects tolerated a 3-minute simulated reentry, attaining 16.5G peak. Subjects were supported in a nylon net couch. Using a molded plastic Mercury couch, subjects achieved 25G peak during 40 seconds. Stoll and Mosely (1958) found that chimpanzees in the fully supine position could endure 40G for 60 seconds, but vascular damage occurred at 40G in semisupine and semiprone positions.

Recovery and landing deceleration: Stapp showed (1955, 1958) that the survival limit for front-to-back deceleration force in lightly anesthetized chimpanzees optimally restrained by nylon webbing was reached at 237G peak with 11,250G per sec rate of onset and 0.35-second total exposure. Persistent injury was found at about 5,000G per sec rate of onset, 135G peak, and 0.35 second; transient injury occurred at 60G with rate of onset higher than 5,000G per second in transverse direction. Decelerations less than 40G with rate of onset lower than

600G per second and total duration below 0.2 second, comparable to deceleration from 120 miles per hour in 19 feet, can be survived with no persistent injury by adequately restrained subjects seated facing either forward or backward to direction of linear force.

162. STAUFFER, F., ERROBO-KNUDSEN, E. O.
Positive acceleration and urine output.
USNSAM Rpt. NM 001.059.02.04, 1953. (Authors' Summary)

The effect of positive radial acceleration on the urinary output has been studied in 10 men. In water-loaded subjects an exposure to positive 3G for 1 minute caused a significant reduction in urinary output and an increase in the specific gravity of the urine.

When these water-loaded subjects were exposed to positive 5G for 1 minute with antiblackout-suit protection, the reduction in urine output was much greater and lasted longer. There was a concomitant greater increase in the specific gravity of the urine. The physiological mechanisms which may be involved have been discussed.

163. STEINER, S. H., MUELLER, G. C. E.
Pulmonary arterial shunting in man during forward acceleration.
J. Appl. Physiol., 16:1081, 1961.

Reduced apical perfusion in the erect position reflects the inability of the right ventricle and pulmonary vascular system to maintain a sufficient hydrostatic column to perfuse these segments. At 8G, without adaptation, the column of blood to be supported would be approximately 30 mm above right ventricular outflow tract; thus, in recumbent position anterior portions of the pulmonary alveolar system would not be perfused. X-ray studies support this concept.

To investigate the matter, the authors determined arterial blood gases during forward acceleration ($+G_x$) at 6 and 9G breathing room air and at 8G breathing 100% oxygen. Arterial saturation fell to 84% at 6G and 75% at 8G. Prebreathing oxygen for 15 minutes prior to acceleration and continuing oxygen inhalation during acceleration partially corrected the undersaturation to 86% at 8G. Recovery was not complete in 3 minutes unless oxygen therapy was used. Whole blood carbon dioxide content was depressed at 6G and 8G on room air. During the recovery period, carbon dioxide content was depressed. The pH was reduced and P_{CO_2} elevated slightly during each acceleration period. Since alveolar ventilation is not significantly diminished during forward acceleration at these G levels, the oxygen desaturation must represent a right-to-left pulmonary shunt. At 6G approximately 40% of cardiac output is shunted and at 8G this is approximately 60%. Oxygen inhalation demonstrates an anatomical shunt of approximately 40% at 8G.

Photomicrographs of anterior-posterior sections of lung taken from a dog killed immediately after forward acceleration at 14G for 10 minutes show in the anterior portion of the lung a marked overexpansion of the alveoli and a striking absence of red cells. The middle region shows relatively normal pulmonary parenchyma with the normal distribution of blood and air. In the dependent regions large areas are totally devoid of patent alveoli and there is massive atelectasis with an increase of blood volume in these areas. There was no pulmonary edema.

Arterial saturation probably falls for a variety of reasons; for example, abnormalities in ratio of ventilation to blood flow, diminished surface for gas exchange, insufficient time for completion of gas transport since rate of perfusion must be increased because of preservation of cardiac output, and perhaps choking of capillaries with blood cells. Reduction in arterial carbon dioxide content and decrease in pH evidently represents replacement by acid metabolites from anaerobic metabolism. Oxygen inhalation corrects the situation where the areas are still perfused, as indicated by better maintenance of the whole blood carbon dioxide content. However, the reduction in carbon dioxide content during the initial recovery period on oxygen indicates that some areas are not receiving adequate perfusion during acceleration, and can only release their metabolites during the recovery period. One subject with a previous history of pneumonia showed the lowest saturation, suggesting that individuals with previous lung disease may have residual involvement accentuating any hypoxia.

164. STEINER, S. H., MUELLER, G. C. E.
Heart rate and forward acceleration.
J. Appl. Physiol., 16:1078, 1961.

Minor differences have been noted in various studies of the heart rate under conditions of forward acceleration. The authors measured the cardiac rate in six human subjects during forward acceleration at $+8G_x$, with rate of onset 0.5G per sec, maintained at peak for 20 seconds. The head position of the subjects was varied, while the rest of the body was maintained in a fixed positon. No change was observed in cardiac rate for the 20-second duration with the head in the neutral position. Heart rate increased 20% with head elevated and decreased 16% with head depressed. The authors note that baroreceptors respond to pressure of static column of blood within the vessels, dependent upon height of column and density of fluid, and at 1G are not sensitive to change in head position of 6 inches. Under 8G the weight of the column is greatly increased and consequently as little as 15 mm height differential between the aorta and the carotid sinus would be sufficient to trigger a compensatory response.

165. STEINER, S. H., MUELLER, G. C. E., CHERNIAK, N. S.
Pulmonary gas transport as influenced by a hypergravitational environment.
J. Appl. Physiol., 16:641, 1961.

Effects on pulmonary gas exchange were measured during forward acceleration ($+G_x$) at 6 and

8G for 3 minutes at a rate of onset of 0.5G per sec. Oxygen uptake was down 16 ml/min at 6G and 71 ml/min at 8G. Carbon dioxide excretion was up 62 ml/min at 6G and 30 ml/min at 8G. Respiratory exchange ratio was up from 0.83 to 1.17 at 6G, and 1.24 at 8G. Minute volume was markedly increased. During recovery periods from 6G an additional 470 ml of oxygen was consumed and 360 ml of carbon dioxide excreted above control values; for 8G, an additional 650 ml of oxygen was consumed and 580 ml of carbon dioxide excreted. Increased metabolic work at the G level studied was small, representing approximately 150 ml/min of oxygen. Respiratory exchange ratio was well above 1, and large oxygen debt was accumulated. Results presented do not reflect steady state but rather mean dynamics of forward acceleration for 3 minutes. Human tolerance was subjectively limited, and many subjects reported fatigue, chest pain, and dyspnea. Heart rate was initially unchanged, but tended to increase toward the end of 3 minutes. Decreased oxygen uptake was not caused by diminution of total pulmonary blood flow.

To determine whether failure of adequate oxygen exchange might be from failure of oxygen diffusing capacity due to mechanical defects, the authors also investigated the effects of exercise on oxygen uptake at 8G in three subjects and found an average increase of 1,500 ml/min during 1 minute of acceleration. Tolerance was limited to 1 minute and the fatigue persisted for several hours. However, limitation in diffusing capacity was obviously not present. The authors suggest that inertial effects of acceleration alter individual ventilation-perfusion relationships throughout the lungs sufficiently to produce a significant shunt. The problem is aggravated by action of inertial forces on greater circulation.

166. STEINER, S. H., MUELLER, G. C. E., TAYLOR, J. L.
Hemodynamic changes during forward acceleration.
Aerospace Med., *31*:907, 1960.

Dogs were accelerated at $+6G_x$, $+10G_x$, and $+14G_x$ for 10-minute durations in the forward facing position. Cardiac output, heart rate, circulation time, blood pressure, respiratory rate, and qualitative appearance of arterial blood were recorded. Changes in cardiac output, namely, 14% decrease at 6G and 23% at 10 and 14G, were considered minimal and found nonsignificant at the 95% level. Changes correspond to decreases in heart rate and increases in circulation time, and indicate an unchanged stroke volume. Blood pressure related to midchest anatomy fell slightly, but probably does not represent a physiologically important alteration in the vital cardiac regulatory area. Since the heart moves posteriorly during forward acceleration, small differences in position of the blood pressure strain gage in relation to actual heart level result in large blood pressure changes which do not reflect true differences in blood pressure. Therefore determination of blood pressure or total peripheral resistance in terms of the neurohumoral regulation of the circulatory system during acceleration, in relation to surface anatomy, is physiologically meaningless. Intravascular or differential manometers, properly G-oriented, should alleviate this. Respiratory frequency was increased, and all arterial blood samples showed qualitative evidence of desaturation.

167. STIEHM, E. R.
Acceleration protection by means of stimulation of reticuloendothelial system.
J. Appl. Physiol., *17*:293, 1962.
NADC-MA-6129.

The author stimulated the reticuloendothelial system (RES) of rats by injections of endotoxin and found that survival time on exposure to 20 positive G was increased in comparison with control. Blockade of the RES was found to reduce tolerance time or maintain it unaffected. RES is closely connected with ability to withstand stress. Resistance to acceleration involves hemodynamics, anatomical compensations for displacement, and ultimately tolerance to cellular anoxia. Thus it has some characteristics of shock, which may be modified by RES stimulation.

Three methods are effective in enhancing acceleration tolerance: Conditioning, hypophysectomy, and RES stimulation. The author suggests a common mechanism. Histologically, conditioning results in the state of RES hypertrophy and adrenal hypertrophy. RES stimulation involves alterations in endogenous steroid metabolism. RES regulates steroid metabolism in part through catabolism of specific hormones, and conversely is affected by steroid hormones. An increase in acceleration tolerance may be mediated through the hypophyseal adrenal axis, probably by way of a specific humoral pattern, approximated closely by hypophysectomy. RES stimulation may affect steroid metabolism to produce the specific hormonal pattern. Similarly, conditioning may produce a favorable pattern, either directly on the adrenals or through the RES. Humoral pattern probably affects cellular metabolism, permitting a readier tolerance of hypoxia. RES stimulation may also increase resistance by other means, such as increased detoxification of metabolic wastes or increase in substances known to play a role in natural resistance (*e.g.*, complement and properdin). As a practical tool in man there are disadvantages such as hyperpyrexia, hypotension, and growth retardation which also occur. A partially purified RES stimulating portion of the lipopolysaccharides has been isolated by Heller but remains to be evaluated.

168. STIEHM, E. R., LYLE, J. M.
Effect of cobalt polycythemia on the acceleration tolerance of the rat.
Aerospace Med., *32*:630, 1961.

On the basis that administration of cobalt induces a polycythemia and consequently increases the avail-

able hemoglobin, the authors administered cobalt to 25 rats, increasing the hemoglobin from 16.9 grams/100 ml to 21.9 grams/100 ml. The tolerance to positive acceleration was measured in 9 animals immediately after cobalt administration, and in 16 animals 10 days after cessation of cobalt, at which time any toxicity has disappeared although the polycythemia persists. No significant difference in tolerance of acceleration was noted in either group as compared with controls.

169. STOLL, A. M.
Human tolerance to positive G as determined by the physiological end-points.
J. Aviat. Med., *27*:356, 1956.

The author noted that the physiological response to acceleration begins with the onset of acceleration and not with the beginning of the plateau. Thus the response is modified by the duration of rise time. This is emphasized by the fact that arterial blood pressure begins to fall immediately upon application and begins to rise sharply immediately upon reduction of acceleration. On this basis, the author analyzed records of 300 experiments on 15 different subjects in terms of total duration, beginning with the beginning of onset and ending with the beginning of offset. Parameters included (1) rise time, (2) time at maximum G, (3) level of maximum G, and (4) rate of acceleration to maximum G. These were compared with respect to (a) duration-intensity, (b) rate of acceleration, and (c) total duration time. Of the 300 experiments, 40 yielded definite physiological end-points. From the end-points a tolerance curve was obtained which also showed the rate of onset. The time at maximum G and the total time were shown to vary inversely with the rate of onset employed and the peak G level obtained. A nomogram was constructed relating rate of onset, acceleration plateau, and duration of grayout. The data showed that lower rates of onset permit a longer time at maximum acceleration before grayout than do higher rates of onset. The author compared date from other sources, and showed a reasonable agreement within the range of her tolerance curves.

170. SUMMERS, L. G., BURROWS, A. A.
Human tracking performance under transverse accelerations.
NASA CR–21, 1964.

Human performance was measured for control display parameters during positive and negative transverse acceleration ("eyeballs in" and "eyeballs out"). Five subjects were given a compensatory tracking task in pitch and roll using a two-axis side arm controller and a CRT (cathode ray tube) attitude display with a moving horizon. Three acceleration levels were used, each of 2-minute duration.

Performance was measured by the integral of absolute error in both the pitch and roll axes. There was a decrement in performance in the pitch axis with increased acceleration, but differences in the roll axis were shown only for one analysis model of two. No differences were recorded in performance due to direction of the acceleration. Physical control characteristics, such as preload and spring constant, did not affect performance or interact with acceleration. Aircraft dynamics affected performance but did not interact with acceleration.

171. USELLER, J. W., ALGRANTI, J. S.
Pilot reaction to high speed rotation.
Aerospace Med., *34*:501, 1963.

Ten experienced pilots were subjected to rotation in the NASA Multi-Axis Test Facility. Pilots were assigned the task of determining and applying corrective torques to counteract rotations induced at rates up to 70 rpm about a resultant axis. The subject's ability to determine the direction in which to apply counter thrust from his instrument display, as well as his agility in timing the thrust application, was recorded. Thrust was counteracted by using a hand controller to actuate jet nozzles mounted on the facility. The pilot performance loss, or error, was determined as a percentage of the total time that he made incorrect torque input to the system. He was penalized for incorrect inputs, not for errors of omission. In general, pilots were able to perform with a performance error ranging from 6.5% to 18%, depending upon the individual. The rotating environment had no measurable influence on their operation or their performance error. Repeated operation produced an improved performance. Motion sickness was relatively infrequent with experienced pilots, although intermittent rotation at rates of 50 rpm or greater for periods longer than 1 hour could induce motion sickness symptoms. Nystagmus was encountered by all subjects tested when acceleration was endured for at least 10 seconds. If the subject concentrated on a centralized area of the instrument panel the effects were reduced.

172. VARENE, P., JACQUEMIN, C.
Existe-t-il un Syndrome Respiratoire Obstructif au Cours des Accélérations Transverses?
Revue de Médécine Aéronautique, *2*:51, 1961.

Having previously demonstrated the occurrence of respiratory obstruction during negative pressure breathing, the authors compared the effects of negative pressure breathing with transverse acceleration (up to $7G_x$), using as subjects five healthy men and one asthmatic. With the normal subjects, they showed statistically that respiratory obstruction occurs above 6G. With the asthmatic subject a marked increase in expiratory resistance was observed with the onset of acceleration. Any respiratory obstruction that occurs in the normal man under transverse G is minor and latent.

173. WARRICK, M. J., LUND, D. W.
Effect of moderate positive acceleration on ability to read aircraft type instrument dials.
WADC–MR–694–10, 1946.

Using a somewhat artificial dial reading test, the authors exposed 34 pilots to accelerations of 3G and 1½G, for total durations, including onset and offset of 4 minutes, and measured the accuracy of dial reading. No physiological problems or subjective impressions were noted. Results showed that pilots attempted to read as many dials at 3G as at 1½G. At 3G, 24% of readings were erroneous as compared with 18% at 1½G.

174. WATSON, J. F., CHERNIACK, N. S.
Effect of positive pressure breathing on the respiratory mechanics and tolerance to forward acceleration.
Aerospace Med., *33*:583, 1962.
WADC–ASD–TR–61–398

The authors refer to previous studies of forward acceleration which showed a proportionate decrease in all lung volumes with the exception of residual volume. This was associated with a fall in midpulmonary position and a decrease in functional residual capacity and maximal breathing capacity. Respiratory frequency increased linearly with increasing G. Intrapulmonic pressure increased for constant volume, shifting the relaxation pressure-volume curve progressively to the right. This resulted in increase in elastic work of breathing and in oxygen consumption. Thus, effects of forward acceleration are similar to those of negative pressure breathing or breathing ambient air under water.

Previous work by Armstrong showed that effects of forward acceleration were relieved by positive pressure breathing. The authors studied the effects of positive pressure breathing on respiratory mechanics and time tolerance to forward acceleration in 12 normal subjects. They found an improvement, particularly in the expiratory reserve volume, which resulted in a return of pulmonary midposition to above normal and an increase in the functional residual capacity. A 67% increase in mean time tolerance to forward acceleration occurred. Previous studies showed that intrapulmonic pressure increases 5 mm Hg per 1G increment in acceleration for constant lung volume. Thus at $+6G_x$ and $+8G_x$, the necessary positive pressure should be 25 and 35 mm Hg, respectively. These pressures, however, were not tolerable since increasing pressure above 3 mm Hg per G resulted in increased chest pain and expiratory resistance. The cause is unknown. The authors found 2 to 3 mm Hg per G the best approximation for proper pressure. Other inequalities of perfusion, diffusion, and ventilation are probably present in addition and require investigation. The primary reason for discontinuing the runs, with or without the pressure breathing, was the occurrence of dyspnea. One subject experienced hemoptysis with rales at both bases immediately postrun, with rapid clearing.

175. WATSON, J. F., CHERNIACK, N. S., ZECHMAN, F. W.
Respiratory mechanics during forward acceleration.
J. Clin. Invest., *39*:1737, 1960

To determine the similarity between forward acceleration and negative pressure breathing the authors exposed four males to $+G_x$ acceleration at levels of 1, 2, 3, and 4G. They measured vital capacity, expiratory reserve, inspiratory reserve, tidal air, residual volume, and static relation pressure-volume curves. Results are shown in figures and tables in the text.

Lung volumes, with the exception of the residual volume, decreased more or less proportionately. Intrapulmonic pressure increases for a constant volume, displacing relaxation pressure-volume curve progressively downward to the right. If compliance is measured from end-expiratory position at each G level and over a range of 5 mm Hg, static total pulmonary compliance becomes progressively smaller as acceleration is increased. Total work of breathing is approximately doubled from 1 to 4G, the increase being entirely in the "elastic" work component, with no change in nonelastic work. Total dynamic pulmonary compliance decreased during acceleration from 1 to 4G.

It has been shown that respiratory defect during transverse acceleration is primarily restrictive since maximal breathing capacity and 0.5 second vital capacity decrease less rapidly than does total lung capacity. Also, oxygen consumption increases. Thus, forward acceleration applies an added load upon muscles of respiration, decreasing volumes and increasing work, without evidence of bronchial obstruction. Nevertheless intrapulmonic pressure increases, implying that the pump is working against additional resistance, approximately 15 mm Hg at 4G. The authors believe this force is a constant related to acceleration and term it P_G. During acceleration, relaxation pressure at each G is greater than resting relaxation pressure by P_G. Since static compliance for each G over the same volume is the same, there is no change in overall elastic properties. If measured at end-expiratory position, static compliance progressively diminishes, the result of the fall in functional residual capacity and pulmonary midposition. Dynamic compliance decreases similarly.

At 4G, relaxation pressure even at zero lung volume is positive. The reason is obscure. The authors suggest that blood pools into the pulmonary circuit upon relaxation after forced expiration, or that during forced expiration under acceleration air is trapped within alveoli, and then released on relaxation. Frequency increases linearly up to at least

12G. All lung volumes decrease, expiratory reserve volume by the largest percentage, implying a downward shift in midpulmonary position and decrease in FRC. Residual volume does not significantly change, and thus becomes a larger percentage of total lung capacity. The significance of this in altering gas exchange remains to be evaluated. Results support the hypothesis that forward acceleration resembles negative pressure breathing. Inequalities of perfusion, diffusion, and ventilation may, however, still be present.

176. WATSON, J. F., RAPP, R. M.
Effect of forward acceleration on the renal function.
J. Appl. Physiol., *17*:413, 1962.
WADD–ASD–TR–61–375.

On the basis that forward acceleration resembles negative pressure breathing and that diuresis is associated with negative pressure breathing, the authors exposed six normal human subjects to $+4G_x$ for 10 minutes in a highly controlled protocol and investigated renal hemodynamics, electrolyte excretion, and water clearance. Forward acceleration produced slight increase in glomerular filtration rate and effective renal plasma flow during and after stress. After acceleration, there was a 20- to 35-minute lag before appearance of an increase in urine volume and water clearance. Changes in water excretion were transient. No change was observed in one subject who was exposed to a mock stress. The authors suggested that acceleration, like negative pressure breathing, may induce an increase in the intrathoracic blood volume, inhibiting release of antidiuretic hormone via a nonosmotic volume-sensitive receptor mechanism located within the thoracic vascular space, probably within the left atrium.

177. WEBB, M. G., GRAY, R. F.
A new method of protection against the effects of acceleration on the cardiovascular system.
Am. J. Cardiol., *6*:1070, 1960.

During World War II the Germans experimented unsuccessfully with a water suit for protection against G. Franks developed a successful water suit during the same period, but it was not adopted because of the superiority of the pneumatic suit in cost, weight, and comfort.

Bondurant et al. in 1958 experimented with complete submersion in an open tank. The subjects used an aqualung to maintain respiration and provide air pressure necessary to prevent a decrease in chest volume due to high water pressure developed under acceleration. It was concluded that the best orientation for the subject was a semirecumbent position with back elevated 35° from horizontal. The highest acceleration sustained was 14G and the end point was chest pain of uncertain origin.

In 1958 Webb and Gray reported results of tests in an open tank with subjects sitting upright submerged in water to eye level and holding their breath. A level of 16G was attained without blackout and without chest pain. The end point was inability to prevent air from being squeezed from the chest.

The authors describe a total-water-immersion G capsule. In three runs with three subjects, one subject was exposed to accelerations of 31G for a period of 5 seconds. One subject terminated at 28G from frontal sinus hemorrhage, and the third subject terminated at 26G from anxiety. All subjects had a few petechiae on nose and lips. One subject had severe pain in left great toe, possibly from a small embolus following 26G run.

The authors comment on the difficulties encountered in the gas-filled spaces, where the gradient of fluid pressure external to the space is opposed by a constant gas pressure. The only place where air pressure and opposing fluid pressure would be equal would be in a plane about midpoint of the greatest dimension of the space, perpendicular to the axis of the resultant acceleration vector. This theoretically should be at some level on the chest in an open system, but in practice it would appear to be at the suprasternal notch. The explanation of this is not known. The position to which the plane of equal pressures in the respiratory system shifted as a result of rotation of the capsule in centrifuge runs is unknown, as are the pressures within the vessels in the chest and the acceleration level at which rupture can be expected. Other dangerous unknowns concern the onset of embolism from small air bubbles entering the circulatory system. A benefit of the system is the buoyancy of the body by the water, which permits easy movement and use of the extremities during high acceleration exposures.

178. WEISS, H. S., EDELBERG, R., CHARLAND, P. V., ROSENBAUM, J. I.
Animal and human reactions to rapid tumbling.
J. Aviat. Med., *25*:5, 1954.

After ejection, tumbling of man and seat may occur at rates exceeding 180 rpm. Rates as high as 240 rpm have been reported. To investigate these effects, the authors exposed animals and human subjects to simple spinning on a horizontal turntable, 8 feet in diameter, capable of speeds up to 200 rpm. The center of rotation was placed at heart or at hips. Recordings included blood pressure, respiratory movement, ECG, and acceleration.

Dogs were anesthetized. In dogs spun with a center of rotation through the heart, signs of circulatory collapse and severe hypoxia were observed at rates of rotation higher than 140 to 150 rpm. With center of rotation at the hips there is less interference with circulation, but the animals displayed severe edema and hemorrhage in the head region.

Human spins with center of rotation at the heart were limited to 125 rpm, and with center at hips to 90 rpm, because of pain and petechiae or hemorrhage

in the head region. Circulatory impairment at these speeds was not serious. Conjunctival petechiae in humans occurred at intensity-durations ranging from 3 seconds at 90 rpm to 2 minutes at 50 rpm with the center of rotation at the iliac crest, and from 25 to 30 rpm higher with the center of rotation at the heart. At those same levels, blood pressure and ECG were within normal limits and adequate performance was possible.

179. WEISS, H. S., EDELBERG, R., CHARLAND, P. V., ROSENBAUM, J. I.
The physiology of simple tumbling.
WADC–TR–53–139, Part 2, 1954.

In order to assess the tolerance limits to head-over-heels rotations or flat spins likely to be encountered in escape from aircraft, human subjects were spun on the horizontal spin-table. The experiments were guided by previous animal investigations, but runs on human subjects were limited to 125 rpm because of the onset of pressure pain in the head or feet. During the spin, the physiological behavior of the humans closely resembled that of the dogs and, on the basis of this, curves for the responses of humans at speeds up to 110 rpm were extrapolated to the level of circulatory failure. By this procedure it was estimated that the borderline of unconsciousness would be reached after 3 to 10 seconds of rotation at 140 rpm and complete unconsciousness after 3 to 10 seconds at 160 rpm with the center of rotation at the heart. The data, together with performance tests and the observation of conjunctival petechiae, were used to determine the time-intensity areas of safe and dangerous rotation.

180. WELLS, J. G., MOREHOUSE, L. E.
Electromyographic study of the effects of various headward accelerative forces upon the pilot's ability to perform standardized pulls on an aircraft control stick.
J. Aviat. Med., *21*:48, 1950.

Using one well-trained subject, electromyographic analysis was made of biceps brachii, triceps brachii, and latissimus dorsi, during 10, 20, 30, 40, and 50 lb pulls on an aircraft control stick under conditions of 1, 2, 3, 4, and 5G, with arm in flexed, intermediate, and extended positions.

Contribution of each muscle changes with arm position. With arm extended, the biceps is dominant; with arm flexed, the triceps is dominant.

Positive G increases the activity of all muscles—least during heavy pulls and when arm is in intermediate position.

181. WHITE, C. S.
Biological tolerance to accelerative forces.
Convair Aeromedical Consultants report, Lovelace Foundation, 1954.

This paper is a review of human tolerance to acceleration as compiled from the literature. It includes definitions of tolerance to acceleration in the $\pm G_z$ and $\pm G_x$ vectors, to radial acceleration, and to impact acceleration. It summarizes the physiological, pathological, and subjective reactions to these accelerations. The occurrence of illusions is discussed, and comments are made on protective maneuvers and devices. Several useful figures are included.

182. WHITE, H. L., ROSEN, I. T., FISCHER, S. S., WOOD, G. H.
The influence of posture on renal activity.
Am. J. Physiol., *78*:185, 1926.

In a comprehensive paper dealing with the influence of the posture on the circulation rate, pulse rate, and output of various urinary constituents, as well as on the blood pressure, the authors describe the effects of posture on the volume of urine. Nine experiments were performed on three subjects in which each subject took no food or water after 10 p.m. on the evening preceding the day of the experiment. At 8 a.m. a subject drank 200 cc of water, voided, and discarded the urine. According to protocol, he then stood or lay for 2 hours, voiding at the end of each 2-hour period, and drinking 200 cc of water at the beginning of each period. Each experiment was continued for four consecutive 2-hour periods. In each case the volume output of urine was found to be much greater in the recumbent position than in the standing position over the corresponding 2-hour duration.

183. WHITE, W. J.
Variations in absolute visual threshold during accelerative stress.
WADC–TR–60–34, 1960.

The effects of moderate acceleration on the absolute thresholds of foveal (cone) and peripheral (rod) vision were measured. This experiment shows that accelerative stress has a consistent and progressive effect on visual performance, the effect being proportional to the magnitude of the positive acceleration.

184. WHITE, W. J., JORVE, W. R.
The effects of gravitational stress upon visual acuity.
WADC–TR–56–247, 1956. (Authors' Summary)

It was the purpose of this study to determine the relationship between increased gravitational force and visual acuity when the factor of reduced cerebral circulation is minimized by the use of protective measures known to ameliorate the gross visual symptoms associated with G stress. It was found that gravitational stress has a significant and progressive effect upon visual acuity. Hypotheses are advanced to account for the difference in visual performance during gravitational stress.

185. WHITE, W. J., MONTY, R. A.
Vision and unusual gravitational forces.
Human Factors, *5*:239, 1963. (Authors' Summary)

The report reviews and evaluates the research and observations pertaining to the effects upon human vision of unusual gravitational force, which will be encountered in space flight operations. Increased G and null gravity are the major topics of discussion. Within each topic the gross and qualitative changes in vision are discussed first. Quantitative and analytic studies are next reviewed, ranging from the application of the threshold methods of psychophysics to the performance of flying tasks such as dial reading. Operation experience of the astronauts is included. Techniques for ameliorating the undesirable effects of acceleration and null gravity are summarized.

186. WHITE, W. J., RILEY, M. B.
Effects of positive acceleration on the relation between illumination and instrument reading. WADC–TR–58–332, 1958. (Authors' Summary)

This study concerns the manner in which the accuracy of quantitative scale readings varies as a function of illumination and acceleration. The following basic findings resulted from an analysis of the data from this experiment:

1. At the higher levels of instrument illumination, increasing acceleration and decreasing luminance produce relatively small increase in reading errors.
2. At marginal levels of illumination, acceleration and luminance interact to produce a relatively large increase in error.
3. Intensity of illumination can compensate for the decline in visual performance at stress levels above 1G.

187. WILSON, R. C., BRYAN, G. L., GREEN, G. A., WILLMORTH, N. E., CANFIELD, A. A., WARREN, N. D.
Aftereffects of intermittent positive radial acceleration.
J. Aviat. Med., *22*:509–517, 1951.

Twenty-six subjects were exposed to seven 1-minute trials at $3G_z$, with an 8-minute rest between trials. A control group was exposed to $1\frac{1}{2}G_z$ to offset rotation effects. A test battery given immediately before and after total G exposure consisted of color naming, arithmetic, steadiness, tapping, number ranking, and word separation. Little significant effect was observed, except that improvement in color naming and steadiness by the experimental group was significantly less than that of the control group.

188. WILSON, R. C., CANFIELD, A. A.
The effects of increased positive radial acceleration upon pupillary response.
In Warren, N. D., Bryan, D. L., Wilmorth, N. E., Wilson, R. C., and Svenson, D. W., Psychological Research on the Human Centrifuge: Final Report. Tech. Rept. N6–ori–77, Task Order 3, Dept. of Psychology, Univ. of Southern California, Los Angeles, 1951.

189. WOOD, E. H., LAMBERT, E. H.
Some factors which influence the protections afforded by pneumatic anti-G suits.
J. Aviat. Med., *23*:218, 1952.

This paper represents the first definitive statement on pneumatic anti-G suits and refers to work carried out from 1943 to 1946 at the Mayo Clinic. Inflation of anti-G suit at 1G produces initial rapid increase in arterial pressure. This is followed almost immediately by abrupt decrease in the heart rate, probably due to a depressor reflex originating principally in the carotid and aortic areas, although other mechanisms in thorax and abdomen may be involved. Cardiac irregularities may occur with slowing of heart rate. Arterial pressure may also be reduced toward normal. The bradycardia product may be abolished by tetraethylammonium chloride. This indicates that the effect is mediated by the autonomic nervous system. The same pattern occurs with inflation of the suit under positive acceleration at 5.5G. With inflation, arterial pressure rises above normal value at heart level but falls below normal value at head level. Since pressure in carotid sinus is not elevated, slowing of heart rate does not occur. In fact, an increase may occur but the maximum increase is not as great as it is at 4.6G without suit.

An analysis of the contribution of portions of the suit show that the whole suit gives protection varying from 1 to 2G; leg bladders only give protection of about 0.2G. Abdominal bladders without leg bladders protect to about 0.6G, preventing blackout but allowing grayout. Leg and abdominal bladders protect to about 1.2G and maintain clear vision. Arm cuffs on upper arms alone produce hardly measurable protection, but arm cuffs with full suit afford a high protection of at least $2\frac{1}{2}G$.

Straining will raise suit protection by about 1G. The valsalva maneuver will produce an increase in arterial pressure followed by a decrease, due to the fact that venous return to thorax is retarded and cardiac output declines. When valsalva is performed with G suit, the secondary marked fall of pressure observed does not occur. Venous return from abdomen is not stopped, because G suit increases intra-abdominal pressure to above intrathoracic pressure. The M–1 maneuver affords protection against blackout of about 2.5G, when properly performed. When combined with G suit, the effect is less than a simple addition of the independent effects, perhaps because the increase of intra-abdominal pressure is common to both procedures. Pressure breathing combined with G suit will increase the protective value. The combination of G suit and crouching is greater than the sum of the protective values of each, partly because the increase in arterial pressure at heart level produced by the G suit represents a greater increase in G tolerance when the vertical heart-brain distance is short as in crouching.

190. WOOD, E. H., LAMBERT, E. H., BALDES, E. J., CODE, C. F.
Effects of acceleration in relation to aviation.
Federation Proc., *5*:327, 1946.
(Abstract from STL–9990–6302–KU–000)

The paper discusses in some detail the following subjects: Force as a factor in man's environment; some of the circumstances in which man experiences acceleration in aviation; the human centrifuge; effects of centrifugal force on man; and the development of antiblackout suits.

191. WOOD, E. H., LINDBERG, E. F., CODE, C. F., BALDES, E. J.
Photoelectric earpiece recordings and other physiologic variables as objective methods of measuring the increase in tolerance to headward acceleration ($+G_x$) produced by partial immersion in water.
WADD–AMRL–TDR–63–106, 1963. (Authors' Summary)

The protection against the effects of headward acceleration afforded the human by immersion in water to the level of the xyphoid and to the third rib at the sternum has been assayed in 15 trained centrifuge subjects. Variations in ear opacity, ear opacity pulse, heart rate, respiration, and reaction times to auditory and visual stimuli were recorded continuously during 15-second exposures to acceleration while the subject was seated in a steel tub mounted in the cockpit of the Mayo centrifuge. The level of acceleration was increased by increments of $+0.5G_z$ to $+1.0G_z$ until complete loss of vision (blackout) was produced when the tub was empty (control) and when filled with water to the level of the xyphoid and also to the third rib at the sternum. A total of 200 centrifuge exposures to accelerations ranging from $+2.0G_z$ to $+9.0G_z$ were carried out.

No systematic alterations in the general pattern, characterized by a period of failure during the first 5 to 10 seconds followed by cardiovascular compensation and recovery from visual symptoms during the latter part of the exposure, were observed during immersion in water. The decrements in ear opacity associated with the various degrees of visual impairment were closely similar; however, the decrements in ear opacity pulse and increments in heart rate were significantly less during immersion in water than when in air. The average values for protection afforded by immersion in water to the third rib against loss of vision and the above three objective variables were 1.8 ± 0.1, 2.7 ± 0.2 and $2.9 \pm 0.2G_z$, respectively. It is believed that at head level a higher blood pressure is required to maintain vision during immersion in water than when no external pressure is applied to the lower part of the body, and that the protection afforded to blood pressure at head level and to maintenance of consciousness is greater than the protection afforded to vision.

192. WOOD, E. H., NOLAN, A. C., DONALD, D. E., EDMUNDOWICS, A. C., MARSHALL, H. W.
Technics for measurement of intrapleural and pericardial pressures in dogs studied without thoracotomy and methods for their application to study of intrathoracic pressure relationships during exposure to forward acceleration ($+G_x$).
WADD–AMRL–TDR–63–107, 1963. (Authors' Summary)

Pleural pressures were recorded simultaneously from the ventral and dorsal regions of the thorax using fluid-filled catheters inserted through the chest wall via No. 16 needle using an air-tight technic. Pressures were referenced to the catheter tip levels determined by anterior-posterior and lateral roentgenograms taken prior to and after a series of 1- to 3-minute exposures of eight anesthetized dogs to accelerations of 2, 4, and $6G_x$ (supine horizontal and 15° head-up and head-down positions).

The negativity of intrapleural pressure in the ventral thorax was uniformly increased during exposures while intrapleural pressure in the dorsal thorax became positive. These changes are believed to result from the increase in weight of the lungs and other intrathoracic elements during acceleration and would be compatible with an average specific gravity of the thoracic contents of about 0.5 since the increase in gradient between the dorsal and ventral recording sites averaged about 0.5 cm H_2O per cm of vertical distance between the sites per G to which the animal was exposed. Esophageal and pericardial pressures were similar or somewhat less negative than the intrapleural pressures at the same horizontal plane in the thorax. All dogs showed decrease in arterial oxygen saturation during exposure to $6G_x$ when breathing air or 99.6% oxygen similar to those previously observed in normal human subjects. Collapse of alveoli and consequent arterial-venous pulmonary shunting of blood appears to be the most likely mechanism for the arterial desaturation observed.

193. WOOD, E. H., NOLAN, A. C., DONALD, D. E., CRONIN, L.
Influence of acceleration on pulmonary physiology.
Federation Proc., *22*:1024, 1963. (Authors' Summary)

This is an outline of only some of the effects of acceleration on respiratory physiology. No attempt has been made to cover the considerable effects of acceleration on pulmonary ventilation and other aspects of lung function which have been well studied. If the impression has been created that accelerations in the ranges currently encountered in the launch and reentry phases of space flight may have strikingly deleterious effects on lung function, this is correct. It has been demonstrated, however, that man

can tolerate such accelerations on the centrifuge without apparent serious decrement in his ability to perform flight maneuvers, and the excellent performance of Russian and American astronauts during all phases of orbital and suborbital space flight is a matter of record. It appears, therefore, that from a practical viewpoint, protection of astronauts against these pulmonary effects of acceleration may not be necessary.

194. WOOD, E. H., NOLAN, A. C., MARSHALL, H. W., CRONIN, L., SUTTERER, W. F.
Decreases in arterial oxygen saturation as an indicator of the stress imposed on the cardiorespiratory system by forward acceleration ($+G_x$).
WADD–AMRL TDR–63–104, 1963. (Authors' Summary)

Progressive decreases in arterial oxygen saturation with increasing degrees of forward acceleration have been demonstrated in eight normal subjects. At 5.6G, arterial oxygen saturation began to decrease from control value of 97% after 130 seconds of exposure.

When breathing 99.6% oxygen this decrease was prevented or its onset delayed and its magnitude reduced, 93% being the lowest figure obtained.

The blood oxygen saturation values indicated by the ear oximeter were quite similar in contour and magnitude to those obtained with the cuvette oximeter by continuous photometric analysis directly on arterial blood. The tendency for the decreases in saturation during acceleration to be greater at the ear than those obtained directly from arterial blood is probably related to the decrease in blood flow to the ear, with consequent increased extraction of oxygen from ear blood, which results from the small vector of acceleration away from the head present in this position.

It is postulated that the arterial desaturation is due to flow of pulmonary capillary blood past poorly ventilated or atelectatic alveoli in dependent portions of the lungs, such that large veno-arterial shunts are created. The atelectasis is thought to result from the increased weight of the blood and lung parenchyma caused by acceleration and resulting in increased segmental blood volume and increases in intravascular and extravascular pressures due to hydrostatic effects in the dependent regions of the lungs plus increases in intrapleural pressure in those areas.

Changes compatible with atelectasis were revealed in chest roentgenograms obtained after exposures to 5.6 and 6.4G when 99.6% oxygen was breathed.

Progressive increases in right atrial and intraesophageal pressures with increasing G load were observed. If the changes in intraesophageal pressure under these circumstances were similar to the change in intrathoracic pressure, apparently effective filling pressure of the right atrium was not greatly affected.

Intermittent positive-pressure breathing did not prevent the decrease in arterial oxygen saturation during forward acceleration in the three subjects in which this was studied.

195. WOOD, E. H., SUTTERER, W. F., MARSHALL, H. W., LINDBERG, E. F., HEADLEY, R. N.
Effect of headward and forward accelerations on the cardiovascular system.
WADD–TR–60–634, 1961.

Applying very comprehensive and sophisticated techniques, the authors exposed seven subjects to accelerative stress in the forward and headward positions. Using compact equipment and a technique of sudden injection of indocyanine green into a right atrium, they measured cardiac output, radial artery pressure, ECG, ear opacity, aortic pressure, ear opacity pulse, respiration, right atrial pressure, blood flow through cuvette, centrifuge rpm, and acceleration at chest level. Runs were completed with and without a G suit inflated to 200 mm Hg.

For headward acceleration the average decrease in cardiac output was 7%, 18%, and 22% at 2, 3, and 4G. With G suit inflated the average values were slightly greater but not statistically significant. The greatest relative increase occurred at 2G. Heart rate increased 14%, 35%, and 56% at 2, 3, and 4G; increases were slightly less without suit inflation, and not statistically significant. Mean aortic pressure increased 9%, 21%, and 27% at 2, 3, and 4G. Inflation of the suit increased arterial pressure slightly more and is regarded as basis for protection provided (1G). Systemic vascular resistance increased 17%, 41%, and 59% at 2, 3, and 4G, with somewhat greater increase when suit was inflated. Arterial saturation decreased from 97% at 1G to 93% at 4G, but with suit inflated it decreased from 96% to 89%. These decreases refer to the first 15 seconds before injection of dye; following injection, saturation could not be measured. When duration of exposure was extended from 1 to 10 minutes no further alteration was demonstrable. Exposure to forward acceleration produced little or no change in cardiac output, heart rate, stroke volume, and peripheral resistance. Aortic pressure increased with higher levels of exposure, as did right atrial pressure. A marked decrease in arterial blood saturation also took place. By an experimental technique, all pressures were corrected for change in the base line of the manometer systems under acceleration.

196. WOOD, E. H., SUTTERER, W. F., MARSHALL, H. W., NOLAN, A. C.
Use of the human centrifuge to study circulatory, respiratory and neurologic physiology in normal human beings and a description of an electronic data processing system designed to facilitate these studies.
WADD–AMRL–TDR–63–105, 1963. (Authors' Summary)

Study of the reactions of a system to transient reproducible degrees of stress is a useful means of elucidating the mechanisms of action of the system. Exposures to positive accelerations can be used to produce sudden decreases in arterial pressure at head level of any desired degree down to zero. Study of the reactions of the cardiovascular system induced in this manner to elucidate circulatory physiology has been only partially exploited.

These same maneuvers can be used to produce temporary reproducible degrees of stagnant anoxia of the retina and brain of conscious normal human beings and hence offer a potentially fruitful field for study of the interrelationships of the level of consciousness, electrical activity of the brain and retina, arterial pressure at head level, and blood flow to these areas.

The hydrostatic effects of acceleration cause profound alterations in the ventilation-perfusion ratios in the lungs which are in opposite directions in the dependent and superior portions of the thorax. Temporary obliteration of air-containing alveoli in the dependent portions of the lungs can be procured under the circumstances associated with large arteriovenous shunts in these regions of the pulmonary circulation.

Studies of tolerance of man to various levels and types of acceleration carried out in multiple laboratories in this and other countries have demonstrated that under properly controlled conditions the aforementioned alterations can be produced with safety and without demonstrable permanent sequelae. It is believed that this means of producing temporary severe alterations in the function of various organ systems of intact conscious individuals offers a valuable means for the further elucidation of their physiology. Because of the multiple interrelated variables involved, use of electronic data processing methods seems practically mandatory for full exploitation of the possibilities.

197. ZECHMAN, F. W., CHERNIACK, N. S., HYDE, A. S.
Ventilatory response to forward acceleration.
J. Appl. Physiol., 15:907, 1960.

The authors measured respiratory frequency, tidal volume, minute volume, and nitrogen elimination in 13 male subjects under $+5G_x$, $+8G_x$, and $+12G_x$ for durations of 1 to 2 minutes, and also oxygen consumption before, during, and after exposure to $+5G_x$, $+8G_x$, $+10G_x$, and $+12G_x$. Frequency increased, reaching an average of 39.2 cpm; tidal volume decreased to an average of 318 cc at 12G. Volume of nitrogen eliminated during a 30-second period, breathing oxygen at 12G, was essentially unchanged, suggesting that alveolar ventilation did not decrease. The most prominent effect was an increase in respiratory frequency, which appeared to be linear for the range of acceleration studied. Oxygen consumption increased with increasing forward acceleration, when measured as total additional oxygen consumed plus any oxygen debt during recovery. There is a critical level of ventilation where a further increase results in decreased arterial oxygen and rise in carbon dioxide tension. It is not known whether ventilation at 12G is near such a critical point.

198. ZECHMAN, F., MUELLER, G.
Effect of forward acceleration and negative pressure breathing on pulmonary diffusion.
J. Appl. Physiol., 17:909, 1962.

The authors measured pulmonary gas exchange and diffusion capacity during forward acceleration ($+4G_x$) and also during negative-pressure breathing (-15 mm Hg). Results showed that pulmonary ventilation increased by the same magnitude during negative-pressure breathing and forward acceleration. Ventilation response to negative-pressure breathing was achieved primarily by increase in tidal volume. Ventilation response at 4G was due to rise in frequency. Negative-pressure breathing and forward acceleration altered alveolar gas composition in the same direction. Carbon dioxide production increased similarly with both. Oxygen uptake increased during negative-pressure breathing, but during acceleration oxygen uptake was not significantly changed. Pulmonary diffusion capacity for carbon monoxide was not significantly altered during negative-pressure breathing but decreased significantly during forward acceleration. Desaturation has been suggested as due to perfusion of lung regions rendered atelectatic by reduction in lung volume, or from right-to-left shunting.

Current data on oxygen uptake, diffusion capacity, and arterial saturation suggest that effects of forward acceleration are greater than can be explained by reduction in lung volume alone. Radiographs suggest alteration in distribution of blood flow from front to back along vector of inertial force. This would lead to increased compliance toward anterior regions and decreased compliance posteriorly, tending to create air shunt anteriorly and blood shunt posteriorly. The decreased diffusion observed probably reflects a decrease in area of functional alveoli in contact with functional capillaries rather than change in membrane permeability. Pulmonary edema may contribute.

199. ZECHMAN, F. W., TAYLOR, J.
Respiratory response to forward acceleration compared with chest compression in dogs.
J. Appl. Physiol., 17:410, 1962.

To determine whether the increase in respiratory frequency during forward acceleration is associated with a vagal reflex similar to that in negative-pressure breathing, the author exposed six dogs to $+6G_x$ for 30 seconds, measuring frequency before and after vagal section. Dogs were also exposed to chest com-

pression. Section of the vagi abolished frequency response to cuff inflation but did not alter response to forward acceleration. After carotid denervation (vagi intact) the frequency again increased. With vagal section and carotid denervation, frequency response to forward acceleration was abolished. Results of the carotid series indicated that a hypoxic drive, mediated by a peripheral chemoreceptor, might be involved. The response when vagi were intact is probably due to the fact that chemoreceptors of aortic body are innervated by vagi and were still operative.

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